The Influence of Recreational Ball Hockey Play on Cardiovascular Risk Factors in Sedentary Males

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SUMMARY

Introduction: The prevalence of coronary artery disease (CAD) is ever increasing in western industrialized societies. An individuals overall risk for CAD may be quantified by integrating a number of factors including, but not limited to, cardiorespiratory fitness, body composition, blood lipid profile and blood pressure. It might be expected that interventions aimed at improving any or all of these independent factors might improve an individual's overall risk.

To this end, the influence of standard endurance type exercise on cardiorespiratory fitness, body composition, blood lipids and blood pressure, and by extension the reduction of coronary risk factors, is well documented. On the other hand, interval training (IT) has been shown to provide an extremely powerful stimulus for improving indices of cardiorespiratory function but the influence of this training type on coronary risk factors is unknown. Moreover, the vast majority of studies investigating the effects of IT on fitness have used laboratory type training protocols. As a result of this, the influence of participation in interval-type recreational sports on cardiorespiratory fitness and coronary risk factors is unknown. Aims: The aim of the present study was to evaluate the effectiveness of recreational ball hockey, a sport associated with interval-type activity patterns, on indices of aerobic function and coronary risk factors in sedentary men in the approximate age range of 30 - 60 years. Individual risk factors were compiled into an overall coronary risk factor score using the Framingham Point Scale (FPS). Methods: Twenty-four sedentary males (age range 30 – 60) participated in the study. Subject activity level was assessed apriori using questionnaire responses. All subjects

(experimental and control) were assessed to have been inactive and sedentary prior to participation in the study.

The experimental group (43 ± 3 years; 90 ± 3 kg) (n = 11) participated in one season of recreational ball hockey (our surrogate for IT). Member of this group played a total of 16 games during an 11 week span. During this time, the control group (43 ± 2 years; 89 ± 2 kg) (n = 11) performed no training and continued with their sedentary lifestyle. Prior to and following the ball hockey season, experimental and control subjects were tested for the following variables: 1) cardiorespiratory fitness (as VO2 Max) 2) blood lipid profile 3) body composition 5) waist to hip ratio 6) blood glucose levels and 7) blood pressure. Subject VO2 Max was assessed using the Rockport submaximal walking test on an indoor track. To assess body composition we determined body mass ratio (BMI), % body fat, % lean body mass and waist to hip ratio. The blood lipid profile included high density lipoprotein, low density lipoprotein and total cholesterol levels; in addition, the ratio of total cholesterol to high density was calculated. Blood triglycerides were also assessed. All data were analyzed using independent t - tests and all data are expressed as mean $\pm$ standard error. Statistical significance was accepted at $p \leq 0.05$. Results: Pre-test values for all variables were similar between the experimental and control group. Moreover, although the intervention used in this study was associated with changes in some variables for subjects in the experimental group, subjects in the control group did not exhibit any changes over the same time period.

BODY COMPOSITION: The % body fat of experimental subjects decreased by 4.6 ± 0.5%, from 28.1 $\pm$ 2.6 to 26.9 $\pm$ 2.5 % while that of the control group was unchanged at 22.7 $\pm$ 1.4 and 22.2 $\pm$ 1.3 %. However, lean body mass of experimental and control

subjects did not change at 64.3 ± 1.3 versus 66.1 ± 1.3 kg and 65.5 ± 0.8 versus 64.7 ± 0.8 kg, respectively. In terms of body mass index and waist to hip ratio, neither the experimental nor the control group showed any significant change. Respective values for the waist to hip ratio and body mass index (pre and post) were as follows: 1 ± 0.1 vs 0.9 ± 0.1 (experimental) and 0.9 ± 0.1 versus 0.9 ± 0.1 (controls) while for BMI they were 29 ± 1.4 versus 29 ± 1.2 (experimental) and 26 ± 0.7 vs. 26 ± 0.7 (controls).

CARDIORESPIRATORY FITNESS: In the experimental group, predicted values for absolute VO2 Max increased by 10 ± 3% (i.e. 3.3 ± 0.1 to 3.6 ± 0.1 liters min -1 while that of control subjects did not change (3.4 ± 0.2 and 3.4 ± 0.2 liters min-1). In terms of relative values for VO2 Max, the experimental group increased by 11 ± 2% (37 ± 1.4 to 41 ± 1.4 ml kg-1 min-1) while that of control subjects did not change (41 ± 1.4 and 40 ± 1.4 ml kg-1 min-1).

BLOOD LIPIDS: Compared to pre-test values, post-test values for HDL were decreased by 14 ± 5 % in the experiment group (from 52.4 ± 4.4 to 45.2 ± 4.3 mg dl-1) while HDL data for the control group was unchanged (49.7 ± 3.6 and 48.3 ± 4.1 mg dl-1, respectively. On the other hand, LDL levels did not change for either the experimental or control group (110.2 ± 10.4 versus 112.3 ± 7.1 mg dl-1 and 106.1 ± 11.3 versus 127 ± 15.1 mg dl-1, respectively).

Further, total cholesterol did not change in either the experimental or control group (181.3 ± 8.7 mg dl-1 versus 178.7± 4.9 mg dl-1) and 190.7 ± 12.2 versus 197.1 ± 16.1 mg dl-1, respectively). Similarly, the ratio of TC/HDL did not change for either the experimental or control group (3.8 ± 0.4 versus 4.5 ± 0.5 and 4 ± 0.4 versus 4.2 ± 0.4, respectively). Blood triglyceride levels were also not altered in either the experimental or

control group (100.3 ± 19.6 versus 114.8 ± 15.3 mg dl-1 and 140 ± 23.5 versus 137.3 ± 17.9 mg dl-1, respectively).

BLOOD GLUCOSE: Fasted blood glucose levels did not change in either the experimental or control group. Pre- and post-values for experimental and control groups were 92.5 ± 4.8 versus 93.3 ± 4.3 mg dl-1 and 92.3 ± 11.3 versus 93.2 ± 2.6 mg dl-1, respectively.

BLOOD PRESSURE: No aspect of blood pressure was altered in either the experimental or control group. For example, pre- and post-test systolic blood pressures were 131 ± 2 versus 129 ± 2 mmHg (experimental) and 123 ± 2 and 125 ± 2 mmHg (controls), respectively. Pre- and post-test diastolic blood pressures were 84 ± 2 and 83 ± 2 mmHg (experimental) and 81 ± 1 versus 82 ± 1 mmHg, respectively. Similarly, calculated pulse pressure was not altered in the experimental or control as pre- and post-test values were 47 ± 1 versus 47 ± 2 mm/Hg and 42 ± 2 versus 43 ± 2 mmHg, respectively.

FRAMINGHAM POINT SCORE: The concerted changes reported above produced an increased risk in the Framingham Point Score for the subjects in the experimental group. For example, the pre- and post-test FPS increased from 1.4 ± 0.9 to 2.7 ± 0.7. On the other hand, pre- and post-test scores for the control group were 1.8 ± 1 versus 1.8 ± 0.9. Conclusions: Our data confirms previous studies showing that interval-type exercise is a useful intervention for increasing aerobic fitness. Moreover, the increase in VO2 Max we found in response to limited participation in ball hockey (i.e. 16 games) suggests that recreational sport may help reduce this aspect of coronary risk in previously sedentary individual. On the other hand, our results showing little or no positive change in body

composition, blood lipids or blood pressures suggest that one season of recreational sport in not in of itself a powerful enough stimulus to reduce the overall risk of coronary artery disease. In light of this, it is recommended that, in addition to participation in recreational sport, the performance of regular physical activity is used as an adjunct to provide a more powerful overall stimulus for decreasing coronary risk factors. LIMITATIONS: The increase in the FPS we found for the experimental group, indicative of an increased risk for coronary disease, was largely due to the large decrease in HDL we observed after compared to above one season of ball hockey. In light of the fact that cardiorespiratory fitness was increased and % body fat was decreased, as well as the fact that other parameters such as blood pressure showed positive (but non statistically significant) trends, the possibility that the decrease in HDL showed by our data was anomalous should be considered.

FUTURE DIRECTIONS: The results of this study suggesting that recreational sport may be a potentially useful intervention in the reduction of CAD require to be corroborated by future studies specifically employing 1) more rigorous assessment of fitness and fitness change and 2) more prolonged or frequent participants.

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LIST OF ABBREVIATIONS

Age = person's age in years

SystBP= systolic blood pressure

DiastBP = diastolic blood pressure

PP = pulse pressure (calculated as SystBP – DiastBP)

MAP = mean arterial pressure (calculated as DiastBP + 0.33×SystBP)

Hghtcm = person's height (in centimeters)

Wghtkg = person's weight (in kilograms)

Wstcm = waist measurement (in centimeters)

Hipcm = hip measurement (in centimeters)

Whratio = waist to hip ratio (calculated as Wstcm/Hipcm)

Bodyfat = body fat percentage

BMI = body mass index

TC = total cholesterol (mg/dl)

HDL = HDL cholesterol (mg/dl)

TRG = triglycerides (mg/dl)

LDL = LDL cholesterol (mg/dl)

NHDL = Non-HDL-cholesterol, defined as the difference between total and HDL-cholesterol (TC-HDL) (mg/dl)

TC_HDL = total cholesterol to HDL-cholesterol ratio (calculated as TC/HDL)

GLUMG = plasma glucose (mg/dl)

HRBPM = heart rate (bpm)

time_mins = time needed to walk 1 mile in order to determine VO2 max (minutes)

VO2Max = maximal oxygen capacity

ECW_TBW = extra cellular water / total body water

BMR = Basal Metabolic Rate

FPS = Framingham Point Scores

CRF= Coronary Risk Factors

CRE= Cardio Respiratory Endurance

CVD= Cardio Vascular Disease

Note: Variables with suffix _2 (for example, HDL_2 or VO2Max_2) indicate the results of post laboratory test.

CHAPTER 1: INTRODUCTION

1.1 Modern Lifestyle and Chronic Disease

It is well documented that populations in western industrialized societies are living increasingly sedentary lifestyles. The time period for this change has corresponded with the post-industrial revolution of the 1800's. Indeed, it could be stated that modern living has, in general, re-directed man's activities from physical labor to intellectual labor with a concomitant increase in leisure and decrease in labor (49). It has been hypothesized that the increase in sedentary lifestyle we are experiencing, occurring in a relatively short period (~ 200 years) and accelerating in severity, is a significant factor in the appearance of a great number of chronic diseases (46). Booth (49) suggests that because our genes were designed for surviving in the wild, a sedentary lifestyle produces maladaptive changes that produce a number of adverse effects. These maladaptations may pre-dispose modern man to a host of metabolic and cardiovascular diseases including (but not limited to) coronary artery disease, obesity and type II diabetes. At the other end of the spectrum, epidemiological studies indicate that regular (or chronic) physical activity reduces morbidity and mortality (68, 83, 111, 143, 161). More specifically, physical activity may reduce and/or postpone the inevitable age-related disabilities with the effect that they are compressed into fewer years at the end of life (96). Although the potential for physical activity in general, and regular programmed exercise in particular, to offset or even reverse the negative effects of a sedentary lifestyle has been recognized by many investigators, it is only recently that the interaction between physical activity / inactivity

and differential expression of genes important in metabolic regulation has been suggested (49).

1.2 Coronary Heart Disease and Risk Factors for Active and Sedentary Population

Cardiovascular disease (CVD) includes the development of atherosclerosis and potential clot formation resulting in a myocardial infarction (5). Myocardial infarction (MI) is defined as sudden death of a patch of myocardium resulting from the long-term obstruction of the coronary circulation (140). The initiating event for an MI is often a fatty deposit called an atheroma that partially blocks or completely obstructs a coronary artery (168). Clinically, the cascade of events leading to this potentially fatal event starts with endothelial dysfunction and an inflammatory response that may predispose the individual to atheroma-generation (168). The end-result of this cascade is potentially ventricular fibrillation leading to a fatal arrhythmia (i.e. cardiac arrest) (140). Clinically such cardiovascular diseases leading to myocardial infarction is responsible for approximately half of all deaths in the United States (3). For some individuals in western societies, this pathology may begin in childhood and progress for decades (7), cascading from endothelial dysfunction to subsequent inflammatory response (165). In this scenario the development of coronary artery disease and early mortality or morbidly should perhaps be considered inevitable (49).

Data from epidemiological studies have been vital in identifying key risk factors for the development of CVD (167, 35). Coronary risk factors (CRFs) are defined "as an aspect of personal lifestyle, an environmental exposure or inherited characteristics which, on the

basis of epidemiological evidence is known to be associated with health related conditions considered important to prevent" (117). Some of the risk factors for CVD include high blood pressure, abnormal lipid profile, obesity, inactive lifestyle, atherogenic diet and elevated blood glucose. Exercise is believed to reduce factors associated with CVD by increasing cardiovascular functional capacity and decreasing myocardial oxygen demand at a given workload (168). Exercise also increases the interior diameter of coronary arteries leading to increased coronary blood flow (6), resulting in improved heart contractility and stroke volume (168). Exercise is also known to reduce body weight (109), body fat (155), hypertension (104), and to increase the level of HDL cholesterol, which is cardiac protective (146). It is also responsible for increased insulin sensitivity (15) that is associated with a reduction in CRF. In addition to affecting CRFs, it is noteworthy to mention that exercise in general provides sense of well being and increases level of self confidence which in turn leads to reduction of depression symptoms (168). Although it is clear that exercise plays a significant role, it is important to note that environmental variables such as diet and genetic predisposition also play major roles in controlling factors associated with CVD. An understanding of the complex interplay of all of these components is important in developing strategies for the prevention of CVD (116).

1.2.1 Statement of the Problem

In contrast to physical training in the form of aerobic exercise, the influence of organized or recreational sport on physical fitness and/or coronary risk factors is unknown. Given the advantages of recreational sport over physical training in terms of compliance, the

possibility that organized or recreational sport can replace or at least augment everyday physical activity levels deserves to be quantified.

1.2.2 Purpose of the study

The purpose of the study was to examine the effects of recreational ball hockey exercise on cardiovascular risk factors in previously sedentary men aged 30-60 years. Sedentary people were defined as those who did not practice any leisure time physical activity.

1.2.3 Hypothesis

We hypothesized that one season of recreational ball hockey will reduce risk factors associated with coronary artery disease in previously sedentary males aged 30-60. To this end we hypothesized that one season of recreational/competitive ball hockey would improve one or more of the following variables: 1) diastolic and/or systolic blood pressure 2) VO2 Max 3) blood lipid profile 4) BMR 5) BMI 6) body composition and 7) waist to hip ratio. As a result, it was hypothesized that participation in recreational ball hockey will reduce overall risk of developing CAD.

CHAPTER 2: LITERATURE REVIEW

2.1 The Influence of Physical Activity on Reversing Effects of Sedentary Lifestyle

The levels of chronic physical activity are one of the predictors for the risk of CVD (25). The Activity Profile of American College of Sports Medicine (4) emphasizes the importance of regular physical activity in prevention of CVD. It should be pointed out that many of the studies cited above do not distinguish between physical activity and exercise per se as an intervention for reducing or preventing CVD. Namely, the vital factor may be movement and energy expenditure versus physical training for performance (168). In the study of Tanasescu et al (154), the association between the amount, types, and intensity of exercise in relation to risk of CVD in a large cohort of men was assessed. Over 44,000 male health professionals had their physical activity monitored every two years over a 12-year period. Their activities were classified as jogging, walking, heavy outdoor work, or sports such as tennis, soccer, cycling, weight lifting or swimming. This study found that men who ran for an hour or more per week had a 42% risk reduction compared with men who did not run. They also found that weight training for at least one hour per week reduces chances for CVD by 23% and that rowing at least one hour per week caries an 18% risk reduction of CVD. The authors concluded that physical activity in the form of, running, weight training, and walking was associated with reduced CVD risk. The authors also noted that average exercise intensity was associated with reduced risk independent of the number of metabolic equivalent task

(MET) -hours spent in physical activity. Williams PT (166) compared physical fitness and activity as separate heart disease risk factors. Data of this meta analyses showed that the risk of CVD decreases inversely with increasing percentiles of physical activity. This risk reduction was associated with being more physically active or physically fit. This study concluded that being unfit warrants consideration as a risk factor, distinctly from inactivity. In another meta analysis, Berlin et al (25) provided evidence to confirm the importance of increased physical activity in preventing CVD. These authors noted that methodologically stronger studies tend to show a larger benefit of physical activity than less well-designed studies. Wanamethee (160) believes that physical activity appears to be a critical factor in both primary and secondary prevention of CVD. This meta analyses of several publications show clear indication of dose-response relationship between overall physical activity and CVD. Fung et al (72) assessed associations between long-term leisure-time physical activity, television watching, and biomarkers of CVD risk among 468 healthy male health professionals. This study concluded that physical inactivity is significantly associated with several biochemical markers of obesity and CVD. This study also suggests the importance of leisure-time physical activity in the prevention of CVD.

2.2 Diabetes and metabolic syndrome

With the increasing prevalence of obesity in North America, the prevalence of diabetes has increased dramatically. The risk for CVD is significantly increased with both type 1 and type 2 diabetes. In patients with diabetes, CVD is the most common cause of death (46). Diabetes is defined as a fasting blood glucose level of 126 mg or above, but the risk

for cardiac death increases continuously from impaired fasting glucose (100 to125 mg/dl) (168). Wanamathee (161) described metabolic index as the sum of obesity, insulin resistance, hypertension, impaired glucose tolerance or diabetes, hyperinsulinemia and dyslipidemia characterized by elevated triglyceride, and low HDL concentrations. All of the features described above are risk factors for atherosclerosis, and thus, metabolic syndrome constituted a significant risk for coronary heart disease (111). The risks for CVD from diabetes with metabolic syndrome are greater than that for simple obesity alone, and therefore, rational approaches to treatment of diabetes and metabolic syndrome are of prime importance.

It appears that insulin resistance is the basis of most if not all of the features of this syndrome. Hyperinsulinemia itself contributes to atherogenicity, and thus, insulin is atherogenic, leading to the CVD and cerebrovascular disease associated with this syndrome. Lakka et al (111) investigated the association of metabolic syndrome and total and cardiovascular disease mortality in middle-aged men. This study investigated 1209 Finnish men, age 42 to 60, at the baseline (1984-1989) who were initially without CVD, cancer, or diabetes. Follow-up continued through December 1998. The metabolic syndrome as defined by the WHO was associated with 2.6 to 3.0 times higher CVD mortality and 1.9 to 2.1 times higher all-cause mortality. The authors concluded that cardiovascular disease and all-cause mortality are increased in men with metabolic syndrome, even in the absence of baseline CVD and diabetes.

2.3 *Interval Training as an Intervention for Reduction of CVD*

Interval training is a form of cardio respiratory training that combines a segment of high intensity work with segments of light to moderate intensity work. (1) It has been a meaningful part of athletic training programs for many years because a variety of recreational activities demand variation in the bursts of movement intensities. This type of training helps to achieve accomplishment of body composition goals. It should be kept in mind that although a certain combination of training factors may primarily challenge a particular energy system, training adaptation to some extent are likely to be seen in all three energy systems (ATP-Pcr, glycolytic system, oxidative system) with interval training program (168).

Interval training has been employed and shown to be an effective exercise modality to improve maximal aerobic power in healthy active populations of men and women (96). Warburton et al (164) found a positive correlation between high intensity interval training and improved health in patients with cardiac disease. The purpose of this study was to examine the underlying benefits of interval training with highly functional patients with CVD. In this study, 14 male subjects were randomly divided in interval training and a traditional aerobic group. The subjects had a previous history of bypass surgery or angioplasty but were in stable condition and highly functional. Both groups training sessions utilized three different types of exercise modes, including combined arm and leg ergometry, treadmill and stair stepping. The difference was that the interval training group's aerobic workout consisted of 2-minute high-intensity bouts at 90% heart rate reserve followed by 2-minute low-intensity bouts at 40% heart rate reserve, while the

aerobic group exercised at 65 % of heart rate reserve. Training sessions were 3 times per week in duration of 30 minutes each. Perhaps the most notable finding of this study was the significantly greater time to exhaustion observed in the interval training group. According to the authors of the study, it appears that IT leads to adaptations that allow greater tolerance than continuous aerobic exercise. Consistent with this, Ede Tjonna et al (67) found that participants who performed interval training for sixteen weeks, successfully eliminated signs of metabolic syndrome, when compared to people who performed regular, moderate intensity exercise. This study was the first one to compare the cardiovascular effects of exercise intensity in individuals with metabolic syndrome. Individuals who took part in the study exercised with different intensities but used the same amount of energy in each training session. The oxygen absorption rates increased by 19% with more vigorous exercise when compared to the moderate intensity group. Tjonna (67) speculated that the higher heart rates required for high intensity training were more beneficial for improving endothelial function, suggesting that shorter and harder training sessions may be beneficial for improved fitness and for the reversal and prevention of CVD. Duscha et al (65) studied the influence of exercise on overweight or mildly obese men and women with mild to moderate dyslipidemia, aged 40 to 65 years. The participants were randomized to low-amount/moderate-intensity (LAMI) exercise, consisting of walking 19 km/week at 40% to 55% of peak maximum oxygen consumption (VO2 Max) VO2; low-amount/high-intensity (LAHI) exercise, the equivalent of jogging 19 km/week at 65% to 80% of peak VO2, or high-amount/high-intensity (HAHI) exercise, the equivalent of jogging 32 km/week at 65% to 80% of peak VO2, or a control group that did no exercise. Over seven to nine months of training, all

exercise groups increased their peak VO2 and time to exhaustion, compared with the control group, with the LAHI and HAHI groups having the greatest VO2 improvements. The authors concluded that even exercising at a moderate level could improve aerobic fitness and parameters known to reduce CVD. Notably, none of the participants lost weight during the study, in part because they likely increased appetite and food intake while exercising and put on muscle weight. Tsekouras et al (158) examined basal (VLDL-TG) very low density lipoprotein- triglyceride kinetics in a group of sedentary young men who underwent 2 moths of supervised interval training (3 sessions/wk; running at 60 and 90% of VO2 Max oxygen consumption in 4-min intervals for a total of 32 min) and a nonexercising control group. Each subject completed two stable isotope-labeled tracer infusion studies in the post absorptive state, once before and again after the intervention (48 h after the last exercise bout in the training group). Peak oxygen consumption increased by 18% after training, whereas body weight and body composition were not altered. Fasting plasma VLDL-TG concentration was reduced after training by 28%, and this was due to a reduced hepatic VLDL-TG secretion rate into the circulation. No significant changes were observed in VLDL-TG concentration. Tsekouras concluded that a short period of interval aerobic training lowers the rate of VLDL-TG secretion by the liver in previously sedentary men.

2.4 VO2 Max and Physical Activity

2.4.1 Definition and age factor

The laboratory measure of maximal oxygen consumption (VO2 Max) is defined as the maximum volume of oxygen that by the body can consume during intense, whole-body

exercise, while breathing normoxic air at sea level (4). This volume is expressed as a rate, either liters per minute (liters min^{-1}) (absolute) or milliliters per kilograms of body weight per minute (ml kg^{-1} min^{-1}) (relative) (5). In the general population, VO2 Max reaches a maximum value around the age of 30 years and declines about 10% per decade thereafter (168). It is well known that genetics plays a major role in a person's VO2 Max (168) and may account for up to 25-50% of the variance seen between individuals (5). However, it is unlikely that chronic physical activity elevates or protects VO2 Max above untrained or sedentary values. At rest, individuals are about 5-8% of VO2 Max (10). Resting VO2 Max is sometimes referred to as a metabolic equivalent (MET). Low intensity exercise is typically in the range of 25 - 40% VO2 max (11). A comfortable walking pace is approximately a 25% VO2 Max effort, while a 40% VO2 Max represents a brisk walking pace or light running. 45 - 70% VO2 Max is in the range of moderate intensity and is labeled as the aerobic training zone (168). As intensity of exercise increases, heart rate and oxygen utilization are increased as well. High intensity training results in > 70-75% VO2 Max. Eventually, even at increased intensity, heart rate and oxygen utilization plateau as they have peaked (11), although overall intensity can still increase due to anaerobic energy utilization (107). Intensity then can be assessed as a percentage of maximum aerobic intensity and quantified as a percentage of VO2 Max. Prolonged activity duration at this level is severely limited since the metabolism of carbohydrates required to generate sufficient energy demands induces lactic acid production, and therefore activity can only be sustained for short period (10) (168). Increased levels of lactic acid in the blood eventually reach a threshold resulting in muscle failure (11).

2.4.2 Training

It is possible to increase VO2 max at any age with appropriate training. For example, sedentary people embarking on a program of aerobic training of sufficient duration and intensity are expected to increase maximal oxygen uptake (VO2 Max) by 10 - 20% as well as the time to exhaustion during sub maximal exercise performance (5). There are two ways to enhance VO2 Max: volume and intensity of training (4). Intensity has been proven enhancer of VO2 Max more than volume (90). For VO2 Max improvement to occur, training should be conducted at an intensity of at least 70 percent of VO2 Max (4). Some studies have clearly shown that high intensity interval training improves VO2 Max (164). Other studies indicated significant changes in aerobic capacity and autonomic changes in heart rate (HR) after aerobic training in middle-aged subjects (109). Some of the studies examined the effect of age on physical working capacity (115), decade after age 30. However, if body composition is maintained and physical activity levels are kept constant, the decline in VO2 Max due to aging is only about 5% per decade (115). The function of the cardiovascular system is to pump oxygen and nutrient rich blood to the tissues of our bodies. The heart produces blood flow or cardiac output through its heart rate and its stroke volume (140). Stroke volume represents the amount of blood pushed forward per heartbeat. If cardiac output needs to be increased, it is done by increasing heart rate, increasing stroke volume or both (140). Cardiac output can also be increased by dilating the arteries and decreasing resistance, which is called peripheral vascular resistance (168). Another way to increase cardiac output is to increase the amount of blood returning to the heart (5). Because the heart functions like a sump pump it means that whatever volume is brought into the pump is the volume that is pushed out of the

pump. Thus, if the amount of blood returning to the heart from venous circulation is increased, the amount of blood pumped out of the heart will be increased (Starling's Law of the Heart) (5). Traditional aerobic exercise has been known to produce many positive adaptations. By exercising the heart can strengthen and pump more with each beat leading to increased stroke volume. Also, the heart rate can be increased in times of demand. Proper exercise may also create more pliable blood vessels and hence the heart has to pump against less resistance (168), resulting in lower systemic vascular resistance and lower blood pressure. Cardiac output can be quantified by measuring VO2 Max, which is therefore used as an indirect measure of the efficacy of particular exercises at stimulating the cardiovascular system. (11). In addition, training induced increases in VO2 Max have also been attributed to changes in muscle oxidative capacity/profile. For example, Suominen et al. (153) found that an 11% increase in VO2 Max with 8 weeks of training corresponded most strongly to increases in aerobic metabolism and oxidative enzymes in skeletal muscle.

2.5 VO2 Max and coronary risk factors

Jette et al (96) tried to determine the relation between cardio respiratory fitness, as determined with the Canadian Aerobic Fitness Test (CAFT), and selected risk factors for CVD in a Canadian population. In this cross sectional study subjects were classified as being in the low-fitness, moderate-fitness or high-fitness category according to VO2 Max predicted from performance on the CAFT. A total of 4082 males, age 30 to 59 years participated in the study. This study concluded that higher level of aerobic fitness, as defined by VO2 Max predicted from performance on the CAFT, is associated with a

more favorable CVD risk profile. Anderson et al (27) examined CVD risk factors, physical activity, and fitness in a young population of Denmark, including 86 men and 115 women, 23-27 years of age. A favorable CVD risk profile was related to a higher VO2 Max, but not to time spent on physical activity. Anderson suggested that in this age group intensity must be high enough to have an effect on VO2 Max before a preventive effect is present. Martinez et al (120) investigated association between VO2 Max, rest energy expenditure and CVD in Brazilian military personnel. In this study, thirteen participants aged (37.9 ±8.7 year), were subjected to biochemical exams and the VO2max assessment based on the 12 minutes' Cooper test. This study reported a significant inverse correlation between VO2 Max and levels of C- reactive protein (CRP). CRP is plasma protein produced by the liver and is used mainly as a marker of inflammation. The authors concluded that VO2 Max is associated with CVD risk and suggested that increased VO2 Max levels should be considered in health prevention. Sudhir et al (150) examined the relationship of cardio respiratory fitness, as indicated by VO2 Max with subsequent incidence of stroke. The authors also compared VO2 Max with conventional risk factors as a predictor for future strokes. The authors examined a population of 2000 for a period of 10 years and concluded that low cardio respiratory fitness was associated with an increased risk for any stroke and ischemic stroke. The VO2 Max was one of the strongest predictors of stroke, when compared to other modifiable risk factors.

2.6 Blood Pressure and Physical Activity

The US Department of Health and Public Services describes normal blood pressure as 120-80 mm/Hg. Prehypertesnion, a syndrome thought to precede hypertension is described as 139-89 mm/Hg. Finally, high blood pressure is described as 140-90 mm/Hg or higher (Table1).

Table 1: Blood Pressure. Source: US Department of Health and Public Services

Category	Systolic (top number)		Diastolic (bottom number)
Normal	Less than 120	And	Less than 80
Prehypertension	120–139	Or	80–89
High blood pressure			
Stage 1	140–159	Or	90–99
Stage 2	160 or higher	Or	100 or higher

Following endurance training, resting blood pressure (BP) is generally lowered in people who are borderline or moderately hypertensive before training. Bouchard et al (35) found that systolic BP decreases by 10 mmHg and diastolic BP 8 mmHg in hypertensive

subjects and slightly less in borderline hypertensives. Whelton et al. (163) studied how physical activity is related to the decrease in arterial blood pressure (META analysis). These authors concluded that aerobic exercise decreases both systolic and diastolic blood pressure [(on average, 3. 84 mm/Hg for systolic blood pressure and 2. 58 mm/Hg for diastolic blood pressure)]. This decrease was greater for hypertensive than for normotensive subjects; similarly it was greater for obese compared to non-obese people. Kokkinos (104) studied the effects of regular aerobic exercise on blood pressure in African-Americans with mild hypertension. The exercise program lasted for 16 weeks, with exercise intensity ranging from 60% to 80% of maximal heart rate. These authors demonstrated that the diastolic arterial blood pressure decreased by 5 mm/Hg ($p < 0.002$), while there was no decrease in systolic BP. Kingwell and Jennings (88) studied the effects of physical activity intensity on BP. In an experimental program that lasted four weeks, three groups performed low, moderate and high intensity aerobic exercise corresponding to 50, 65-70 and 80-90% of pre-determined maximal work capacity, respectively. In the low intensity group, the subjects performed one hour of walking five times a week while the medium intensity group cycled for 15 minutes five times a week and the high intensity group cycled for 30 minutes three times per week. Their results showed that moderate intensity biking lead to the greatest decrease in blood pressure (by 5/3 mm/Hg) (systolic/diastolic) but that smaller changes were evident with even low intensity walking (3/2 mmHg) (systolic/diastolic). In contrast, high intensity biking did not lead to any significant changes to blood pressure. Thus, the influence of exercise on blood pressure decreases may occur as a function of both exercise intensity and duration.

On the whole, the studies cited above indicate that recreational aerobic exercise has a positive effect on reducing both systolic and diastolic blood

2.7 Blood lipid profile and physical activity

Several different classes of lipids play important roles in normal physiology. Lipids are an important part of the diet and must be transported to all cells of the body. There is a basal level of lipids in the blood and the concentration increases immediately following a meal. Lipids in the blood are absorbed by liver cells to provide energy for cellular functions. The liver is responsible for providing the proper concentrations of lipids in the blood. (140). Some lipids are utilized by brain cells to synthesize brain and nerve tissue. Excess lipids in the blood are eventually converted into adipose tissue. If lipid levels in the blood become too low, the body synthesizes lipids from other foods, such as carbohydrates, or removes lipids from storage. The body also excretes some lipids in the form of fats, soaps, or fatty acids as a normal component of feces. Lipids may be deposited on the walls of arteries as a partial consequence of their insolubility in the blood. (168). Since lipids are hydrophobic and will not dissolve in the aqueous blood plasma (140). This problem is overcome by complexes called lipoproteins, tiny droplets with a core of cholesterol and triglycerides and coating of proteins and phospholipids (168). The coating not only enables the lipids to remain suspended in the blood but also serves as a recognition marker for cells that absorb it. The complexes are often referred to as serum lipoproteins because their concentrations are expressed in terms of volume of blood serum, not whole blood. A very important lipid is cholesterol, which is present in cell membranes and is a precursor of bile acids and steroid hormones. (140). However, in

addition to its important roles during normal physiology, increased cholesterol levels is thought to be a major etiological factor in CHD. The relationship between serum cholesterol levels and CVD is continuous and graded. Evidence from animal studies, epidemiological studies and clinical trials indicates conclusively that high serum cholesterol is a major risk factor for CVD and that lowering cholesterol reduces the risk (3,8, 152,161). Cholesterol circulates in the plasma in three major sizes of lipoprotein particles. Namely VLDL, LDL, and HDL. Total cholesterol is equal to the sum of these three fractions. CVD is directly and linearly related to the levels of total cholesterol and LDL cholesterol and inversely related to HDL cholesterol (5). The US Department of Health and Public Services provides guidelines to the levels of total, LDL and HDL cholesterol and the risk to CVD The US Department of Health and Public Services describes normal total cholesterol level less than 200 mg/dl, low density lipoprotein cholesterol (LDL) level 100 mg/dl, and high density lipoprotein cholesterol (HDL) level above 40 mg/dl (Table 2).

Table 2: Blood Lipid Profile. Source: US Department of Health and Public Services

Total Cholesterol Level	Total Cholesterol Category
Less than 200 mg/dL	Desirable
200-239 mg/dL	Borderline high
240 mg/dL	High
LDL Cholesterol Level	LDL Cholesterol Level
Less than 100 mg/dL	Optimal
100-129 mg/dL	Near optimal/Above optimal
130-159 mg/dL	Borderline high
160-189 mg/dL	High
190 mg/dL and above	Very high
HDL Cholesterol Level	HDL Cholesterol Category
Less than 40 mg/dL	A major risk factor for heart disease
40-59 mg/dL	The higher, the better
60 mg/dL and above	Considered protective against heart

It is well established that vigorous exercise lowers total blood cholesterol levels (147,164,111). The mechanisms underlying this decrease are complex. Saladin et al (140) suggests that exercise reduces the sensitivity of the right atrium of the heart to blood pressure, resulting in less secretion of atrial natriuretic factor. Consequently, the kidneys excrete less sodium and water, leading to a rise in blood volume. This dilutes lipoproteins in the blood, and the adiposites compensate by producing more lipoprotein lipase. Thus, the adiposites consume more blood triglycerides reducing VLDL particles, which shed some of their cholesterol in the process, and HDL pick up this free cholesterol for removal by the liver. A low level of serum HDL cholesterol is an important predictor of coronary heart disease. Several large epidemiological studies suggest that for each one mg/dl increase in HDL there is a 2 percent decrease in CVD risk in men and 3 percent decrease in women (103, 115,3). Furthermore, a low LDL cholesterol level does not eliminate the risk imparted by a low HDL but a high HDL appears to offset some of the risk of a LDL. HDL plays a critical role in reverse cholesterol transport. In addition, HDL cholesterol may retard aterogenesis through prevention of LDL cholesterol oxidation and monocyte adhesion to endothelial cells and by maintaining endothelial function. There is growing recognition in the medical community that non high density lipoprotein cholesterol (NHDL) also strongly relates to cardiovascular risk (82). NHDL can be calculated by subtracting HDL from total cholesterol. NHDL may be particularly important measure in populations where dislipidemia is characterized by low HDL and elevated triglycerides. NHDL has been shown to correlate with CVD severity and progression. (82) Non-high-density lipoprotein cholesterol (non-HDL) contains all known and potential atherogenic lipid particles. Therefore, non-HDL level is a good

potential predictor of risk for CVD as low-density lipoprotein cholesterol (LDL) (82). Cuy et al (60) tried to determine whether non-HDL level could be useful in predicting CVD mortality by using data from the Lipid Research Clinics Program Follow-up Study. A total of 2406 men and 2056 women aged 40 to 64 years at entry were observed for an average of 19 years, with CVD death as the main outcome measure. Levels of HDL and non-HDL at baseline were significant and strong predictors of CVD death in both sexes. This study has found that non-HDL level is a somewhat better predictor of CVD mortality than LDL level. The authors suggested that the lower NHDL the better and that 130 mg/dl is often a good NHDL goal, based on particular history and risk factors.

Kokkinos et al. (103) studied the influence of aerobic exercise (running) on blood lipid profile in 2,906 healthy, nonsmoking, middle-aged men (mean age: 43 years). The subjects were stratified into six groups based on the mean number of miles run per week (5, 9, 12, 17, and 31). Values for high density lipoprotein (cholesterol) were found to increase by 0.308 mg/dl with each 1-mile increase in running distance. Significantly higher HDL-C levels were observed in the groups that ran 7 or more miles per week compared with the non-runners. Furthermore, mean HDL levels were significantly higher in those who ran 12 or 17 miles per week versus non-runners and those who ran 5 miles per week, and were significantly higher in those who ran 31 miles per week versus all of the other groups. Interestingly, Katzmarik et al. (86) found that changes in blood lipids associated with aerobic exercise training are not related to changes in aerobic fitness but are instead associated with body fatness.

2.8 Framingham Point Scale

Risk assessment and risk factor modification have become essential tools in the management of cardiovascular disease. The Framingham risk prediction model uses total cholesterol along with other major risk factors in estimated the 10 year risk of coronary events in persons without known atherosclerotic CVD (2). The Framingham risk prediction model incorporates HDL and HDL cholesterol of 60 mg/dl or more as a negative risk factor i.e.: it's presence removes one risk factor from the total count when determining a persons major risk factors that modify LDL cholesterol goals. Framingham Study researchers made a great leap forward by inventing precise marker of cardiovascular risk disease. Framingham risk scores furnish 2 ways to estimate relative risk. One compares a given individual's estimated risk with the absolute risk of an individual at low risk, i.e., a person who is largely without risk factors. The other compares a given individual's estimated risk with the risk of an average person of the same age and sex. The latter ratio is commonly used, although it tends to underestimate the preventable component of coronary risk (5). The Framingham charts provide a realistic picture of a given individual's true absolute and relative risks. Therefore, they can be helpful in tailoring a plan for risk factor management. Another potential use of the Framingham charts is patient education and motivation. Patients with low risk scores can be reassured. Those with higher scores should, as a minimum, be counseled to adopt risk-reducing life habits, i.e., smoking cessation, dietary change, weight control, and exercise. It must be emphasized, however, that a low absolute risk, particularly in young adults, does not ensure a lifetime of low risk.

CHAPTER 3: METHODS

3.1 Subject Recruitment

The target population was healthy, sedentary males between the ages 30 and 60 years of age. Sedentary people were defined as those who did not practice any physical activity in leisure time. Rationale for this selection indicated the fact that the male population showed much more interest for ball hockey and was easier to recruit than their female counterparts. Also, the age 30 to 60 has been chosen because it was most likely to provide potential presence of coronary risk factors. Subjects were recruited through newspaper advertisement. Subjects were required to provide letter from family doctor stating that they are healthy in order to participate in this study. Participants were divided into control and experimental groups. The experimental (training) group was formed into a ball hockey team and this team participated in one season of recreational ball hockey (Ball Hockey International, St. Catharine's ON). All subjects were informed of the purpose, methods, and potential risks of the study, including ball hockey play. No prior hockey experience was required. All players played~15 minutes per game and rotated their positions. This study was approved by the Brock Research Ethics Board (REB 07-087- Poleksic). Before testing, an informed consent that meets ethics approval was given and signed by those who met the criteria.

3.2 Testing Protocol

Anthropometric and other laboratory measurements were conducted at Brock University Exercise Physiology Laboratory. The Rockport Test for determination of VO2 Max was conducted on the indoor track of the Walker Complex Sport Facility at Brock University. Subjects in the experimental group reported to the laboratory for pre- and post-season physiological testing. Pre-season testing took place in the last week of May and first week of June, within 2 weeks of the start of the ball hockey season. Post-season testing took place in the third week of August, within 1 week of the conclusion of the ball hockey season. Subjects in the control group reported to the laboratory for physiological testing within the same time frame (May and August, respectively) at the same time as the experimental group. Subjects in control group were advised to continue sedentary lifestyle.

3.3 Testing Procedures

All participants were tested on two occasions at the applied exercise physiology lab at Brock University. The participants were instructed to refrain from exercising, consuming alcohol, and eating or drinking at least 8 hours before testing. Upon arrival to the lab, body composition, anthropometric measurements, VO2 Max, blood pressure and blood samples for determination of blood lipids and cholesterol were taken. The order and protocol of tests was the same for all subjects and was the same for pre- and post-training periods. Both experimental and control subjects were required to fill out questionnaires

regarding dietary and activity habits for the between-testing period and to arrive for laboratory testing in overnight fasting state.

3.4 Anthropometry

After arrival of the subjects into laboratory and after all paperwork was conducted anthropometric measures were taken. In order to measure height, participants were asked to stand straight with no shoes on, with their back against a wall. Mounted to the wall was a stadiometer with a vertical ruler and sliding projection, which was adjusted to rest on the participant's head. Height was measured to the nearest millimeter. The same investigator performed the all anthropometric measures. Waist to hip ratio was measured using tape measure. With participants top clothes off, waist circumference was measured around smallest area of waist, usually just above belly button. Secondly, hip circumference was measured on widest parts of the buttocks. Calculation was obtained by dividing the measure of the waist by this of the hips (6). Percent of body fat and weight were measured by using bioelectrical impedance analysis (BIA) device. Lean body mass was calculated from body fat % and weight of the participants (6). Upon completing anthropometric measures, subjects have proceeded to BIA device. The Biospace In Body 520 Analyzer (135) divides the body into five segments-4 limbs and a trunk and measures the impedance of each segment at multiple frequencies (5,50 and 500kHz). The DSMF-BIA method has a high level of accuracy because of the combination of varying frequencies and segmental analysis. The participants were advised to stand on the instrument bare footed an to hold the bars wide apart. Previously they were advised to

take belts off, any change from the pockets or anything that would compromise accurate recording. The same investigator performed all measurements.

3.5 Blood Pressure

It was recorded by PyMah sphygmomanometer (37), (PyMah Corporation, Flemington, NJ). Participants were comfortably sitting with both feet on the floor and right arm exposed and tight clothing removed. Hands were resting on the table with palm upwards. Large cuff was applied to bare skin, 2 cm above fossa antecubitalis. After finding a pulse at brachial artery toward the inside of the elbow, stethoscope was applied over pulse. Two measures were taken and recorded. The same investigator performed all measurements. Two readings, five minutes apart, were recorded.

3.6 Blood Analysis

In the sitting position and after cleaning the middle finger of the non-dominant hand with an alcohol swab, two small drops of whole blood were collected by finger pinprick utilizing disposable single use lancets. The site of the pinprick was once again cleaned with a fresh alcohol swab and covered with an adhesive bandage. Blood was tested for total cholesterol (TC), high density (HDL), low density (LDL) cholesterol, non high density cholesterol (NHDL) along with triglycerides (TG), glucose and Total Cholesterol/HDL ratio. The blood analysis was done by using the Cholestech GDX and LDX (121), (Hayward, CA, USA), small portable finger pinprick analyzer. This unit is a valid and reliable instrument capable of obtaining results in 5 minutes from one drop of whole blood. The analyzer is a U.S. National Glycohemoglobin Standardization Program (NGSP)-certified instrument for the quantification of blood HbA1C (% of total hemoglobin), which is directly proportional to the concentration of glucose in the blood over the past 2-3 months. The Cholestech LDX meets all U.S. National Cholesterol Education Program (NCEP) guidelines for precision and accuracy regarding measurements (in mg/dL) of total cholesterol (TC), HDL, TG, glucose, TC/HDL ratio, and estimates of LDL and VLDL. The same investigator performed all measurements.

3.7 VO2 Max Testing

The Rockport Walking Test (162) as a form of sub-maximal field test was used to estimate VO2 Max. This test was designed by Rockport Walking Institute in 1986 and is well suited for sedentary individuals since it carries very low risk of adverse effects. The participants were required to walk one mile (1600 meters) as quickly as possible on the

middle lane at the Brock indoor track facility. Prior commencing the walk, participants have performed 5-minutes of light stretching. All of the participants have been wearing appropriate light clothing and running shoes. Participant's heart rate was monitored with polar HR monitors and was recorded immediately upon the completion of 8 laps (1600) meters. Time to complete one mile was recorded with Timex stopwatch. The participants VO2 max was estimated by using the following formula (162):

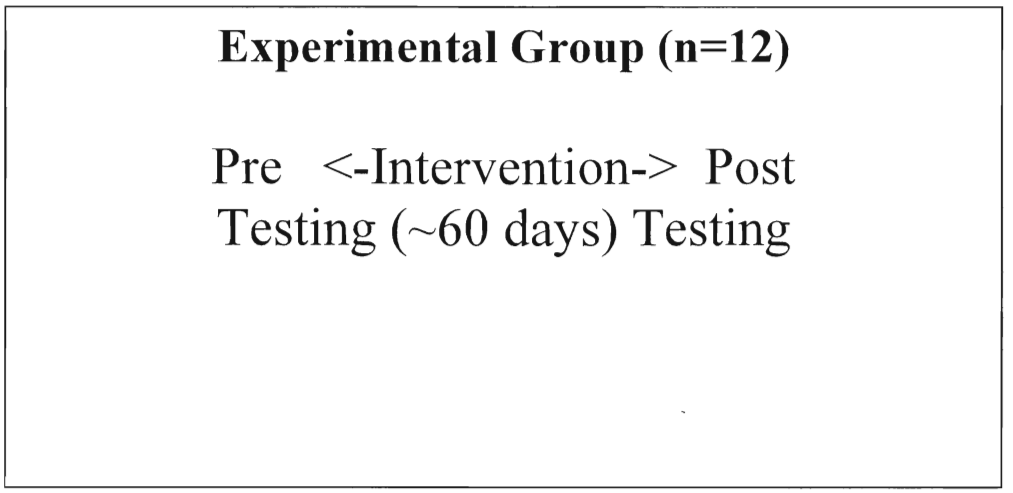
Estimated VO2max ml kg^{-1} min^{-1} = 132.853 – 0.0769(Weight) – 0.3877(Age) + 6.315(Gender) – 3.2649(Time) – 0.1565(HR)

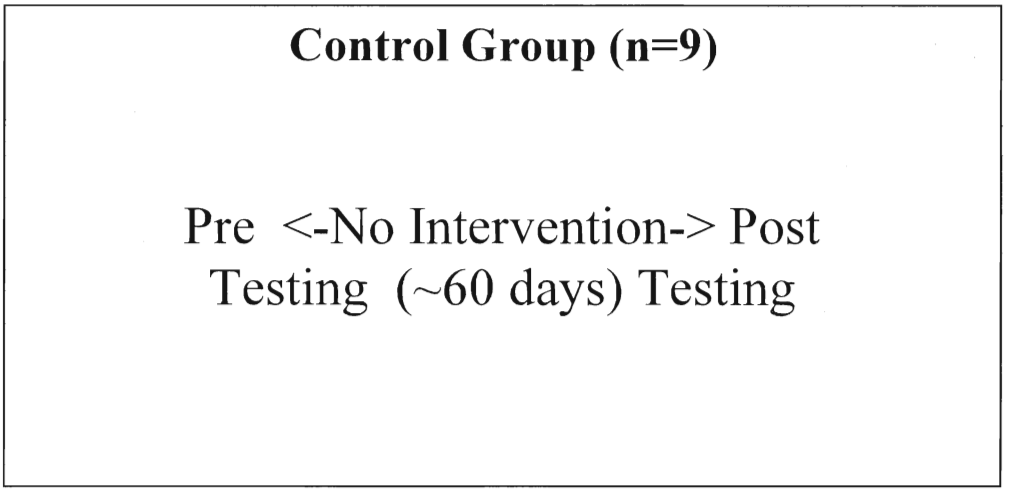
3.8 Questionnaires

Questionnaires (nutrition, PAR-Q, training history, subject screening and medical history, subjective exercise experience scale) were completed prior to laboratory testing by the subjects, with the help of the investigator.

Figure 1

Time Line For Study





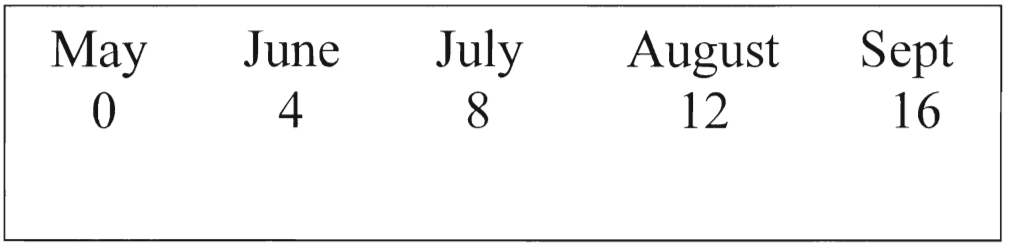
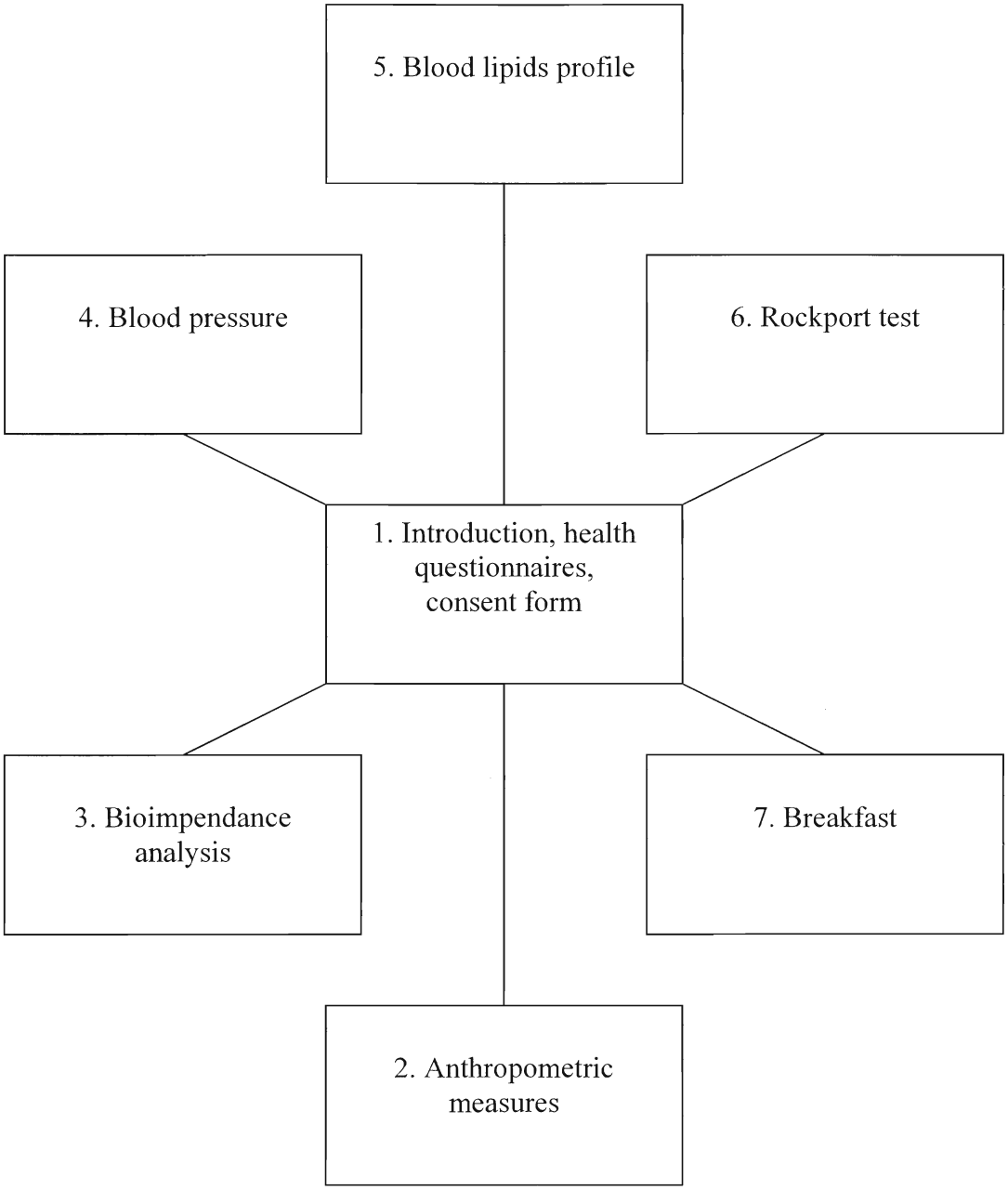


Figure 2

LABORATORY FLOW CHART



3.9 Study Design

This study has utilized a longitudinal design to investigate the effects of recreational ball hockey on cardiovascular risk factors. In between pre- and post-season testing, schedule for experimental group was 16 games of ball hockey in 10 weeks giving a frequency of 1.6 games per week In other words, the schedule planned for 1.6 games per week. But with compliance at 94%, they actually played (1.6*0.94=1.5) games per week per individual. Player attendance was monitored and non-compliance resulted in exclusion of one experimental group subject from the study. Overall, player compliance (i.e. participation in scheduled games) was (15/16*100)=94%. One player was excluded from the study on the basis of non-compliance. However, two subjects were excluded from the study based on medications.

3.9.1 Ball Hockey Game Protocol

All participants were required to arrive 15 minutes prior the game. The participants were instructed to refrain from consuming alcohol with 24 hours of game time and eating at least 4 hours before the game. All participants were equipped in proper uniforms and appropriate safety gear. The game was structured so every participant was guaranteed 15 minutes of playing time. Assessing the average and/or peak intensity of ball hockey game play was beyond the scope of this study. However, players were randomly selected before games and asked to wear either a heart rate monitoring device (Polar) or an actigraf accelerometer.

3.10 Statistical Analyses

Data were analyzed by using the SPSS for Windows (version 16, SPSS Inc., Chicago, IL) statistical software package. T-tests for independent samples were used for evaluating differences among the two study groups (experimental versus control). Paired samples t-tests were utilized to identify the difference in measurements between pre and post laboratory tests. Mean values and standard deviation were used as descriptive statistics. Level of significance was assumed to be $p < 0.05$.

CHAPTER 4: RESULTS

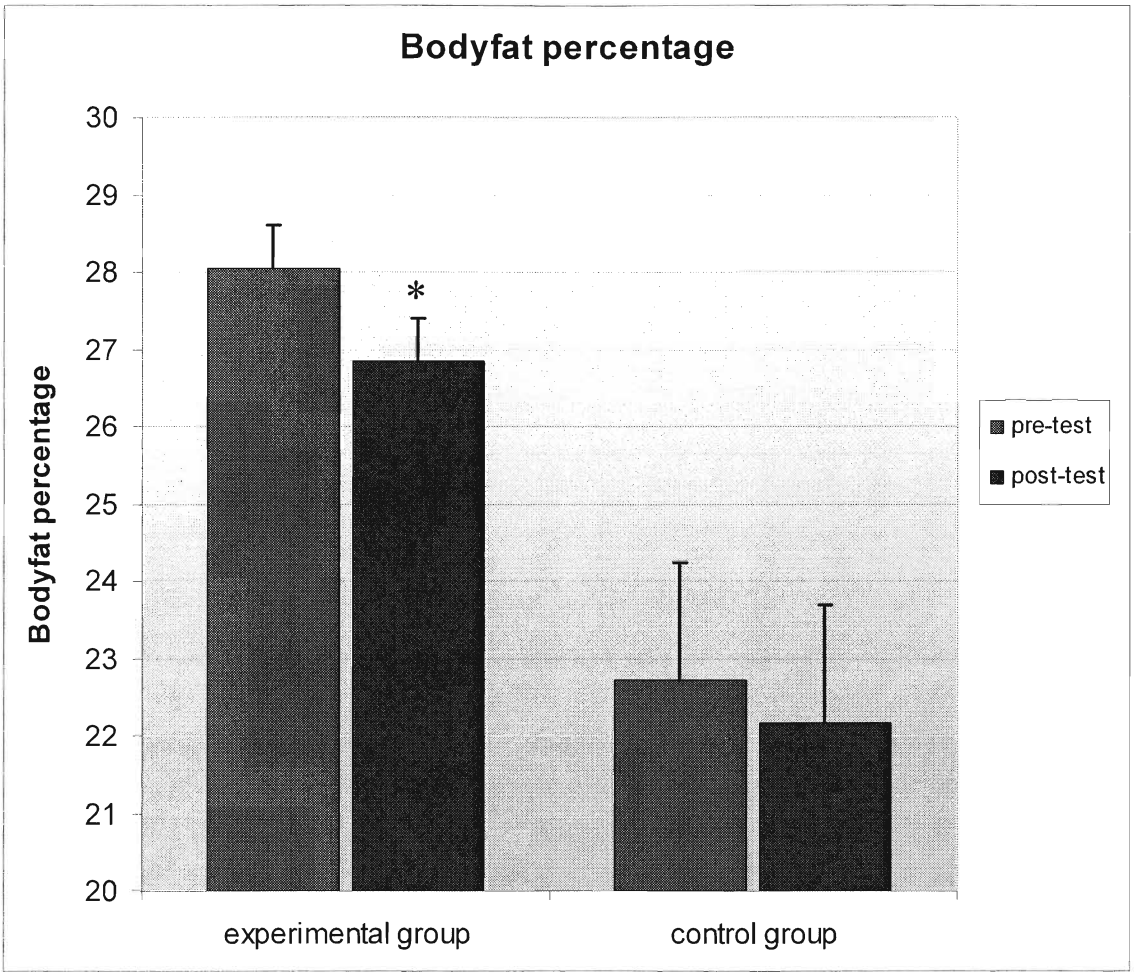
In the current study we investigated the impact of Ball Hockey on Coronary Risk Factors in sedentary males. The result of this work showed an improvement inVO2 Max and body fat percentage in reducing risks for development of coronary disease. Interestingly, however ball hockey was associated with a decrease in HDL and increase in FPS, which are adversely, associated coronary risk factors. The other CRF assessed in this study, including weight, height, BMI, LBM. BP, PP, TC, LDL, NHDL, TC/HDL ratio, and Glucose were unchanged.

4.1 Body composition

Following one season of ball hockey we observed a significant decrease in mean values of body fat from 28.1 ± 2.6 % to 26.9 ± 2.5 % ($P < 0.05$). In contrast, the % body fat of the control group was unchanged at 22.7 ± 1.4 % and 22.2 ± 1.3 % (before and after testing, respectively). These data are summarized in Fig 3. On the other hand, the waist-to-hip ratio for both the control and experimental group did not change. For example, pre- and post-test values for the experimental group were 1 ± 0.1 and 0.9 ± 0.1, respectively, while for the control group pre- and post-values were 0.9 ± 0.1 and 0.9 ± 0.1, respectively (both $P < 0.05$). These data are summarized in Fig. 4. Lean body mass stayed unchanged for both groups. For example, pre- and post-values for the experimental group were 64.3 ± 1.3 kg versus 66.1 ± 1.3 kg respectively, while for the control group were 65.5 ± 0.8 kg versus 64.7 ± 0.8 kg. These data are summarized in Figure 5. Similarly, body mass index for control and experimental groups did not change. For example, pre- and post-values

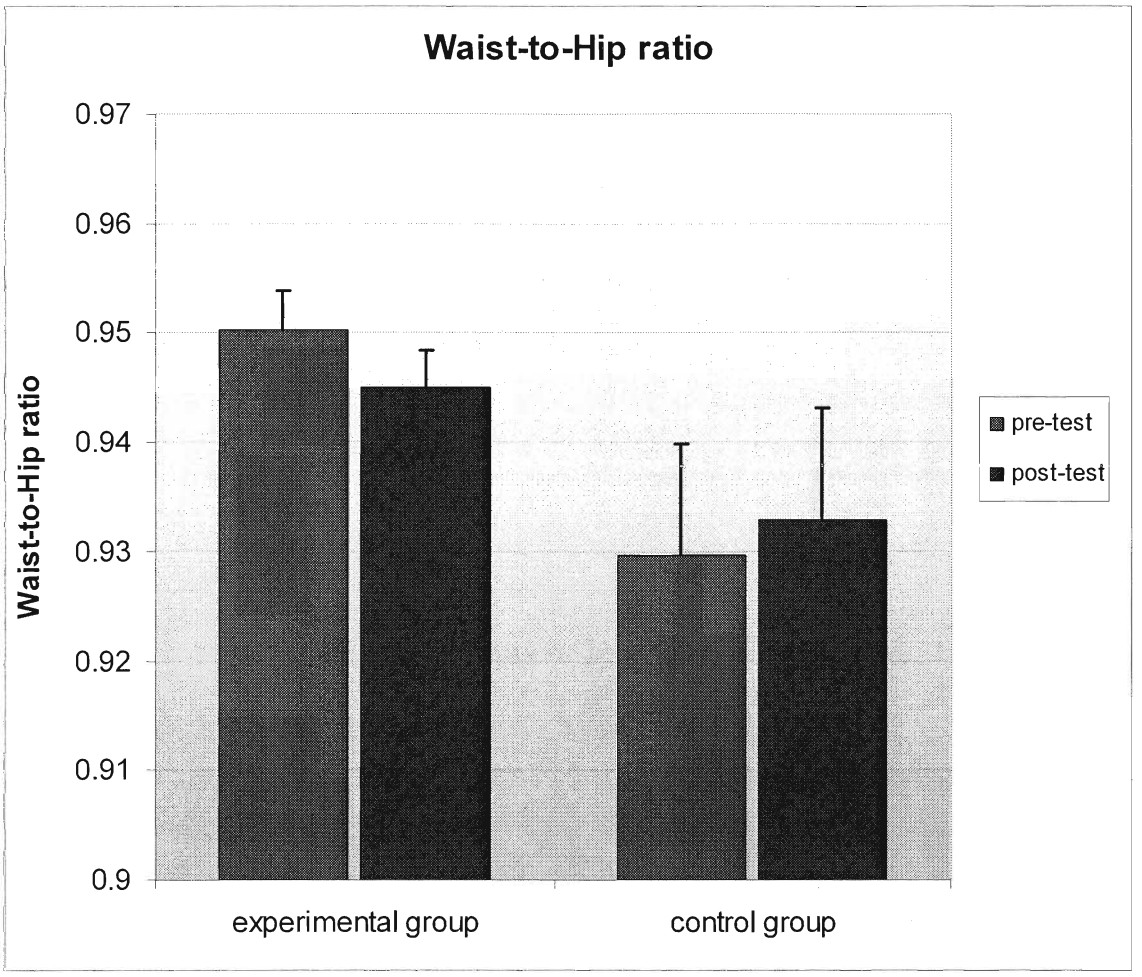
for the experimental group were 29.2 ± 1.4 and 28.9 ± 1.2, respectively. For example, pre- and post-values for the control group were 26.3± 0.7 and 26.2 ± 0.7, respectively. These data are summarized in Fig. 6. The percentage changes of these anthropometric data are summarized in Fig. 7.

Figure 3. Body Fat percentage for control and experimental group (pre- and post- tests).



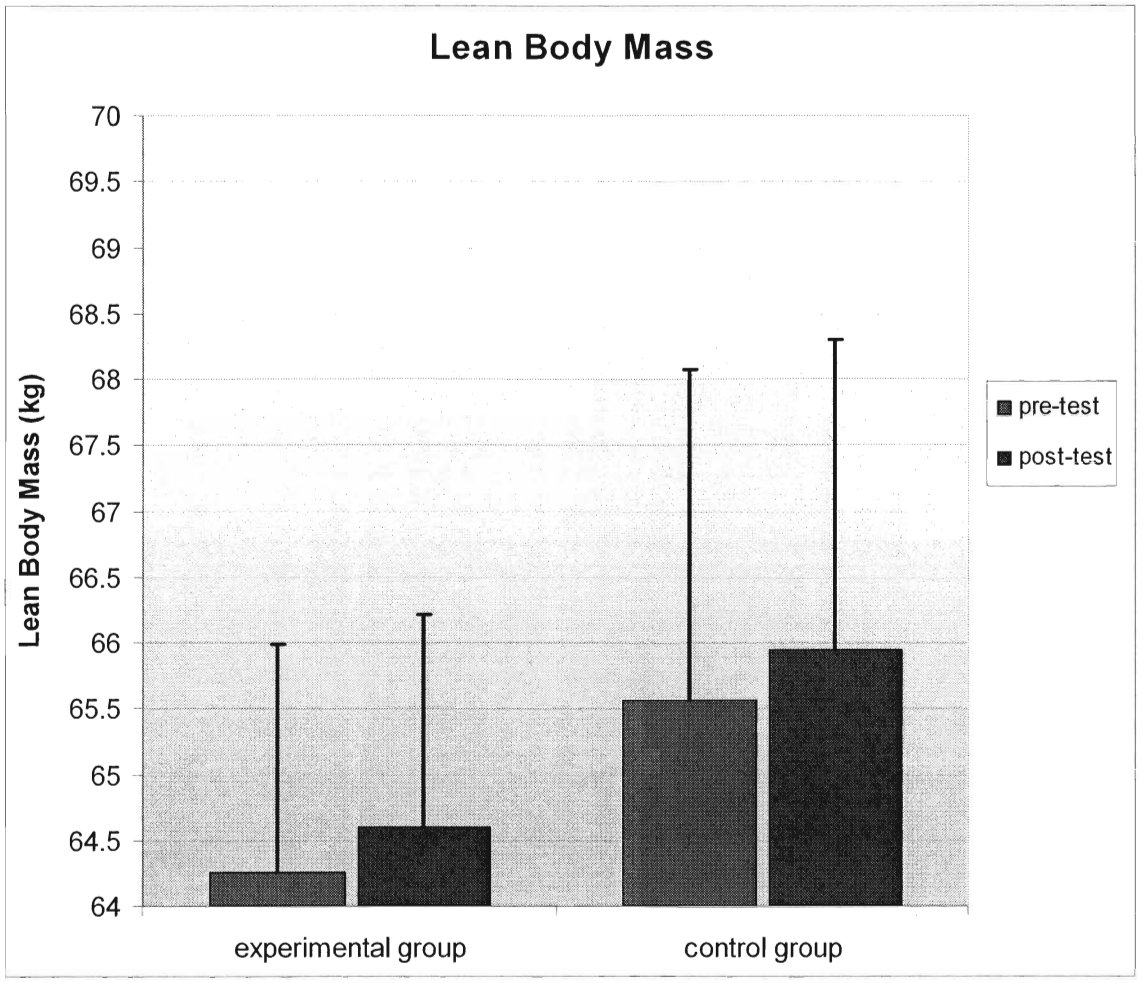
Values are means ± SEM. Asterisk shows statistically significant change ($p < 0.05$).

Figure 4. Waist-to-hip ratio for control and experimental group (pre- and post- tests).



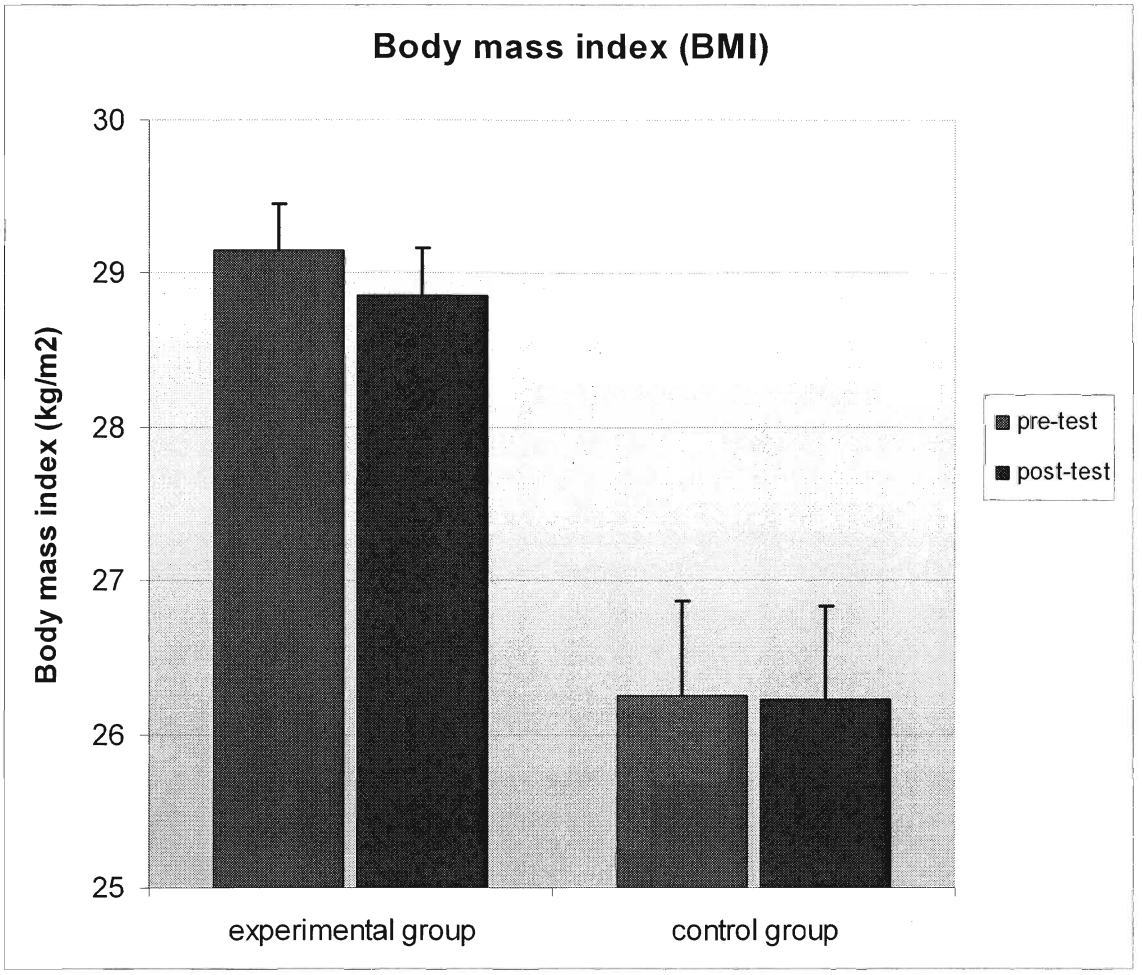
Values are means ± SEM. Asterisk shows statistically significant change ($p < 0.05$).

Figure 5. Lean Body Mass (pre- and post test)



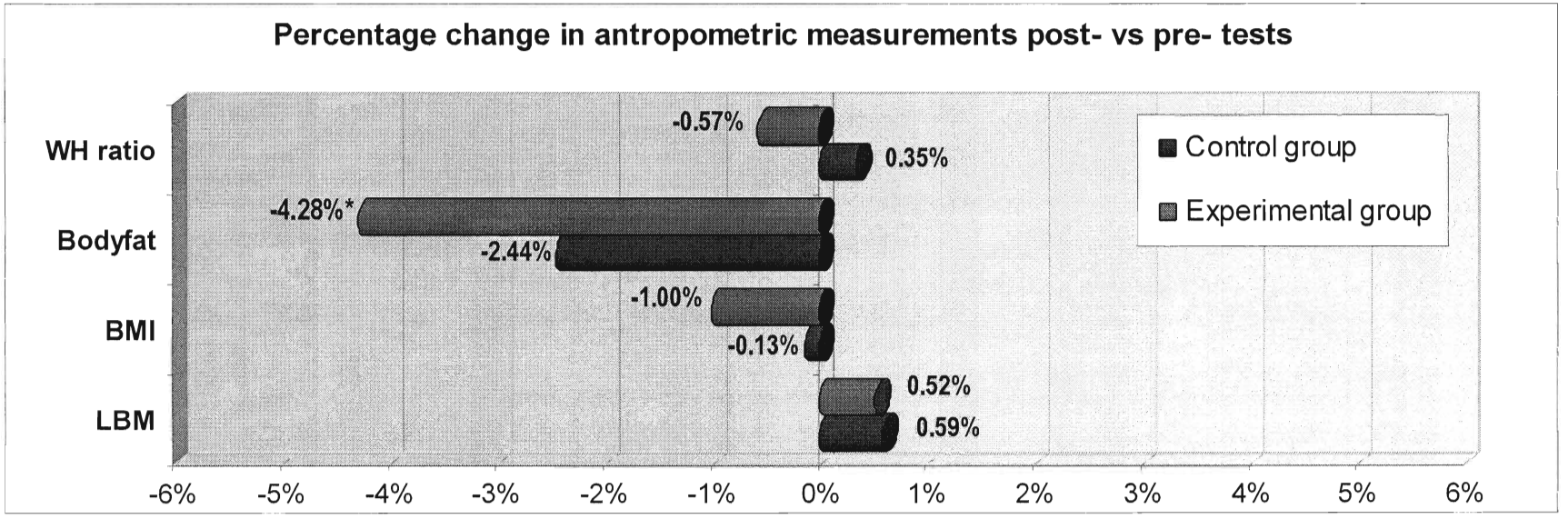
Values are means ± SEM. Asterisk shows statistically significant change ($p < 0.05$).

Figure 6. Body mass index for control and experimental group (pre- and post- tests).



Values are means ± SEM. Asterisk shows statistically significant change ($p < 0.05$).

Figure 7. Percentage change in anthropometric measures for control and experimental groups (pre- and post-test).



Values are means ± SEM. Percentage change in mean values between pre- and post-tests in two separate groups (negative values represent a decrease in post-test versus pre-test). Body fat percentage decreased by 4.3% in the experimental group. Note that no other fluctuations were statistically significantly. Asterisk shows statistically significant change ($p < 0.05$).

4.2 Cardiorespiratory Endurance

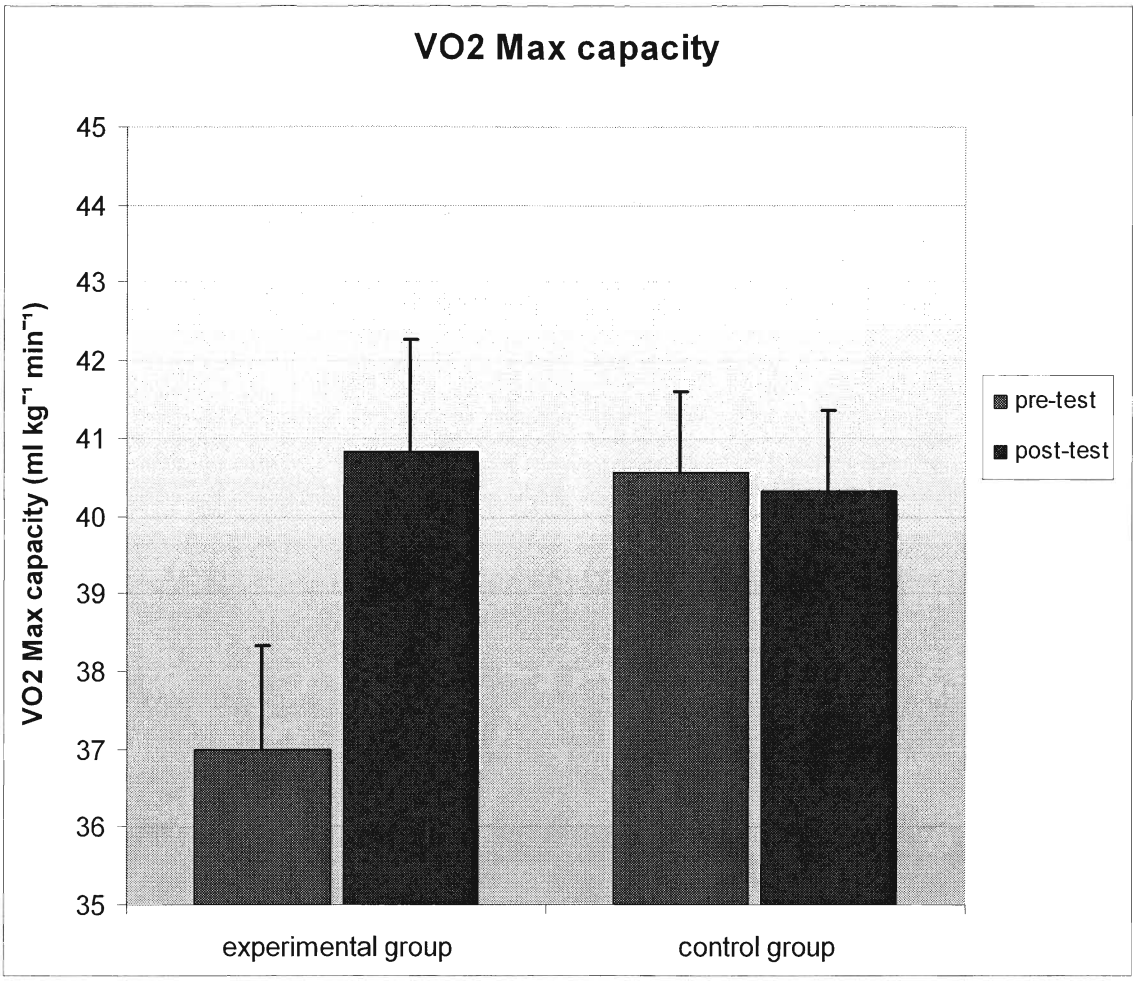
Following one season of ball hockey we observed a significant increase in relative VO2 Max of 10.4 $\pm$ 2.8 %. For example, predicted VO2 Max of experimental subjects increased from 37 $\pm$ 1 to 41 $\pm$ 1 ml kg^{-1} min^{-1} (relative VO2 Max). However, absolute VO2 Max of this group was unchanged at 3.3 $\pm$ 0.13 and 3.6 $\pm$ 0.1 liters min^{-1} (Figures 8 and 9). On the other hand, both the relative and absolute VO2 Max of the control group was unchanged at 41 $\pm$ 1 and 40 $\pm$ 1 ml kg^{-1} min^{-1} and 3.4 $\pm$ 0.2 and 3.4 $\pm$ 0.2 liters min^{-1}, respectively (Figures 8 and 9). Scrutiny of the heart rate data derived from the Rockport Test reveals a reduction in submaximal heart rate during the 1 mile walk (all Rockport Test data variables summarized in Fig. 10), confirming an increase in aerobic function.

4.3 Blood Lipids (HDL, TC, HDL / TC, LDL, TRG)

We observed a significant decrease in mean values of HDL 52.4 $\pm$ 4.4 mg/dl to 45.2 $\pm$ 4.3 mg/dl ($p < 0.05$) in the experimental group. On the other hand, the control group data for HDL was unchanged. As an example, pre- and post –test HDL levels were 49.7 $\pm$ 3.6 and 48.3 $\pm$ 4.1 mg/dl, respectively. These data are summarized in Fig. 11. However, total cholesterol did not change in either the experimental or control group (181.3 $\pm$ 8.7 versus 178.7 $\pm$ 4.9 mg/dl) and 190.7 $\pm$ 12.2 versus 197.1 $\pm$ 16.1 mg/dl, respectively (summarized in Fig.12). The calculated ratio of TC/HDL did not change for either the experimental or control groups were 3.8 $\pm$ 0.4 versus 4.5 $\pm$ 0.5 mg/dl and 4 $\pm$ 0.4 versus 4.2 $\pm$ 0.4 mg/dl, respectively (Fig. 13). In addition, the LDL levels did not change in either the

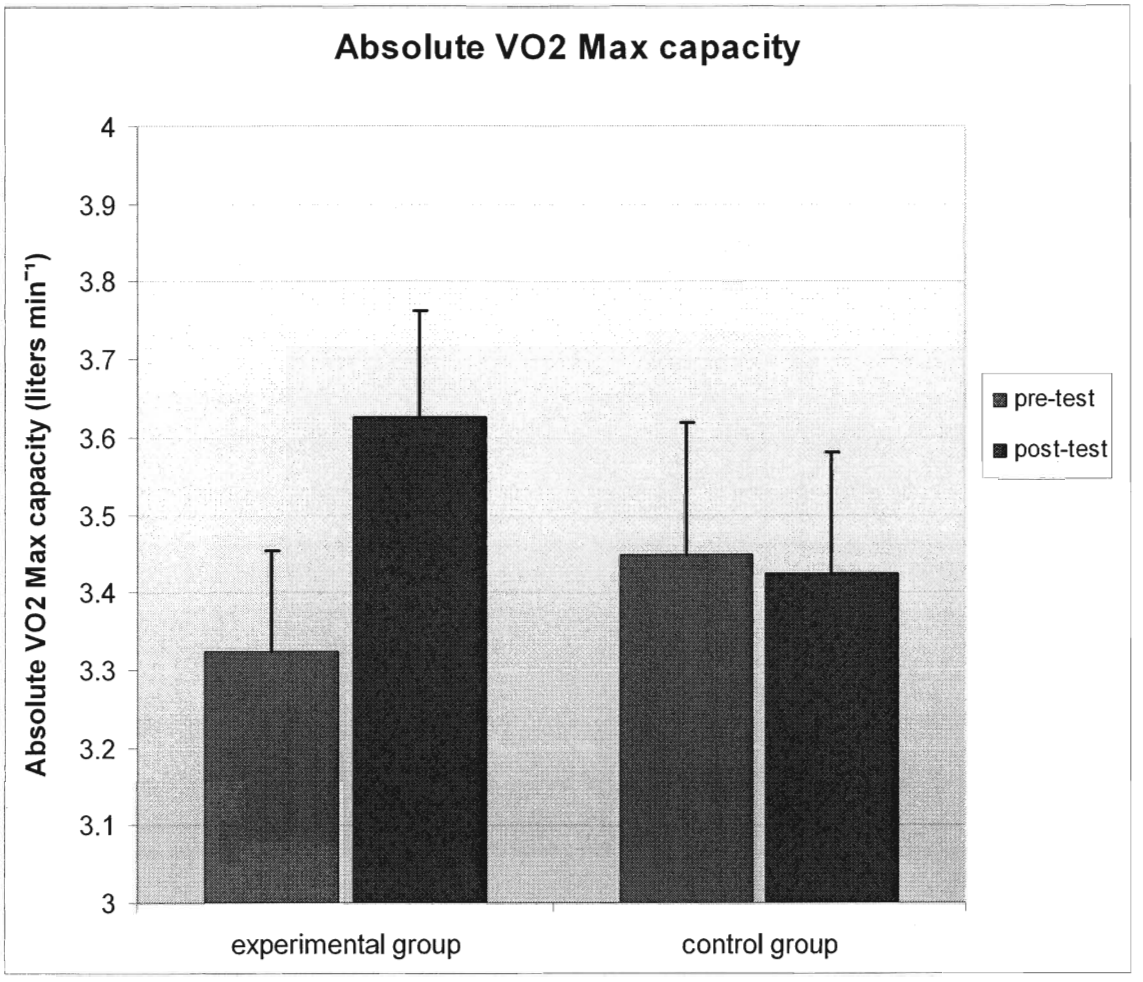
experimental or the control group. For example, pre- and post-test values for the experimental and control groups were 110.2 ± 10.4 versus 112.3 ± 7.1 mg/dl and 106.1 ± 11.3 versus 127 ± 15.1 mg/dl, respectively (Fig. 14).

Figure 8. VO2 Max values for control and experimental groups (pre- and post- tests)



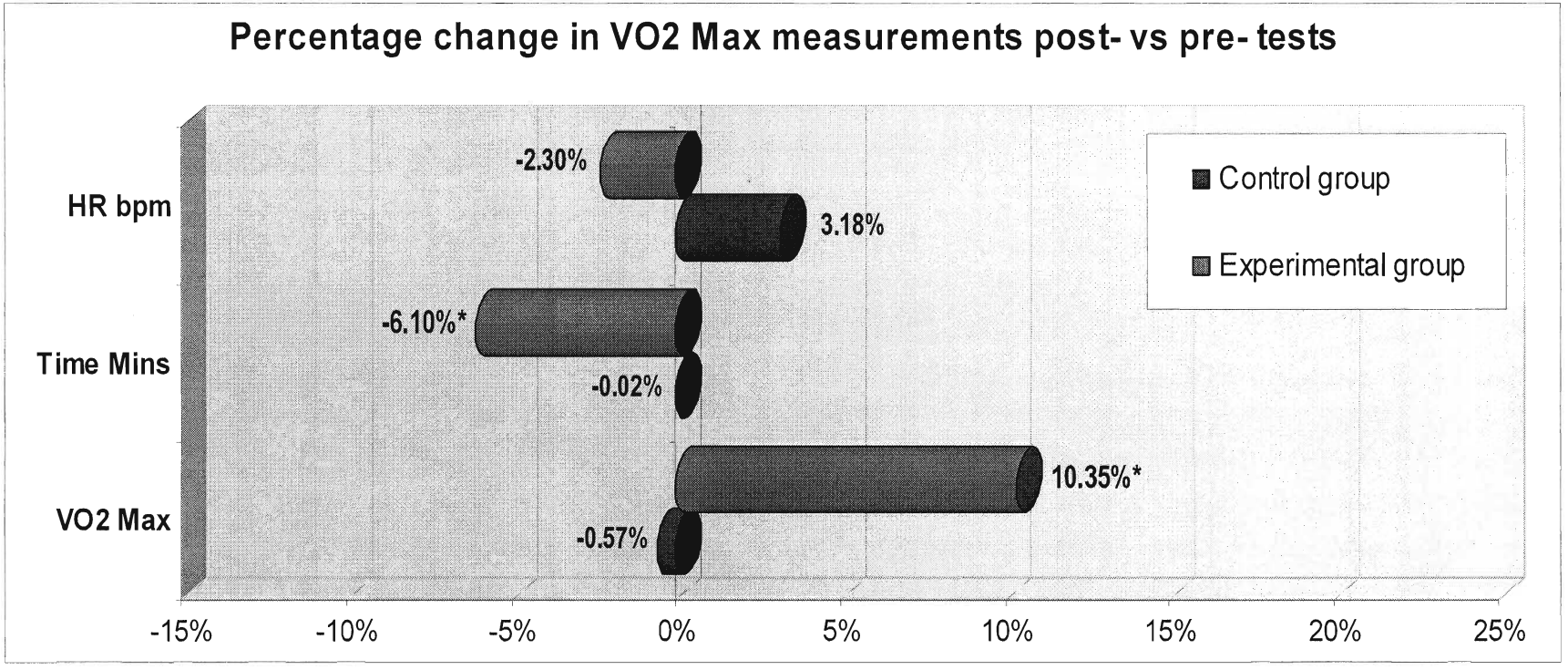
Values are means ± SEM. Asterisk shows statistically significant change ($p < 0.05$).

Figure 9. VO2 Max values for control and experimental groups (pre- and post- tests)



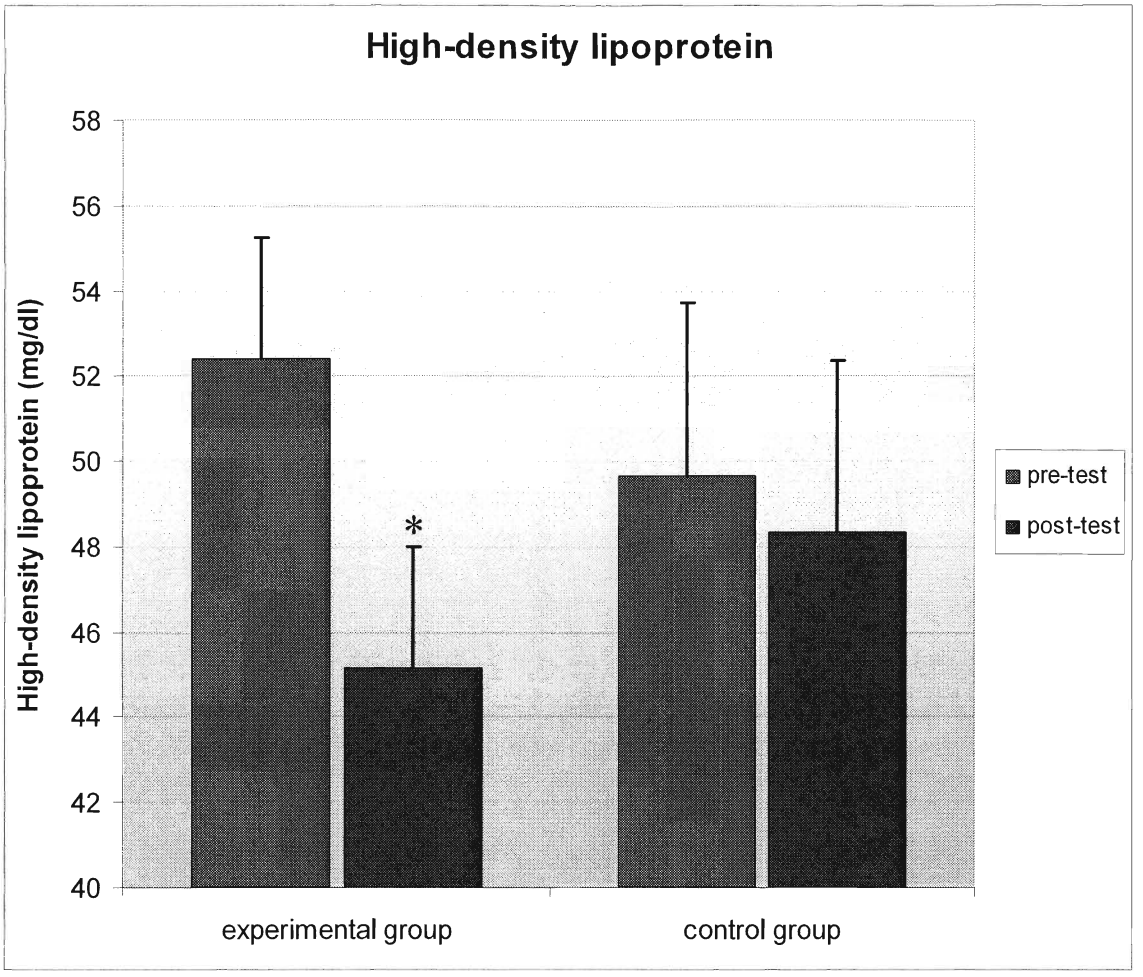
Values are means ± SEM. Asterisk shows statistically significant change ($p < 0.05$)

Figure 10. Percentage change in Rockport Test data for experimental and control groups (pre- and post – test).



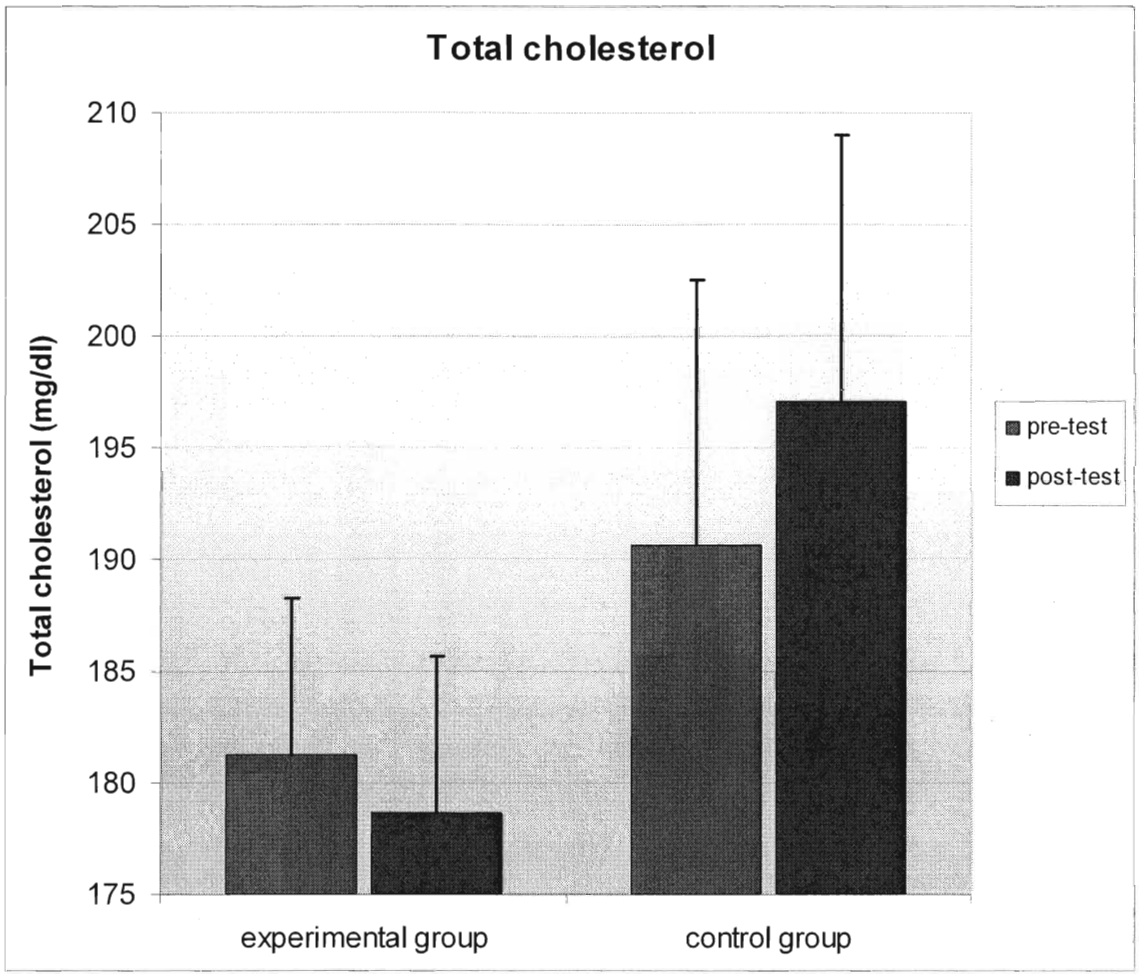
Values are means ± SEM. Asterisk shows statistically significant change ($p < 0.05$).

Figure 11. Values for HDL cholesterol for control and experimental groups (pre- and post- tests).



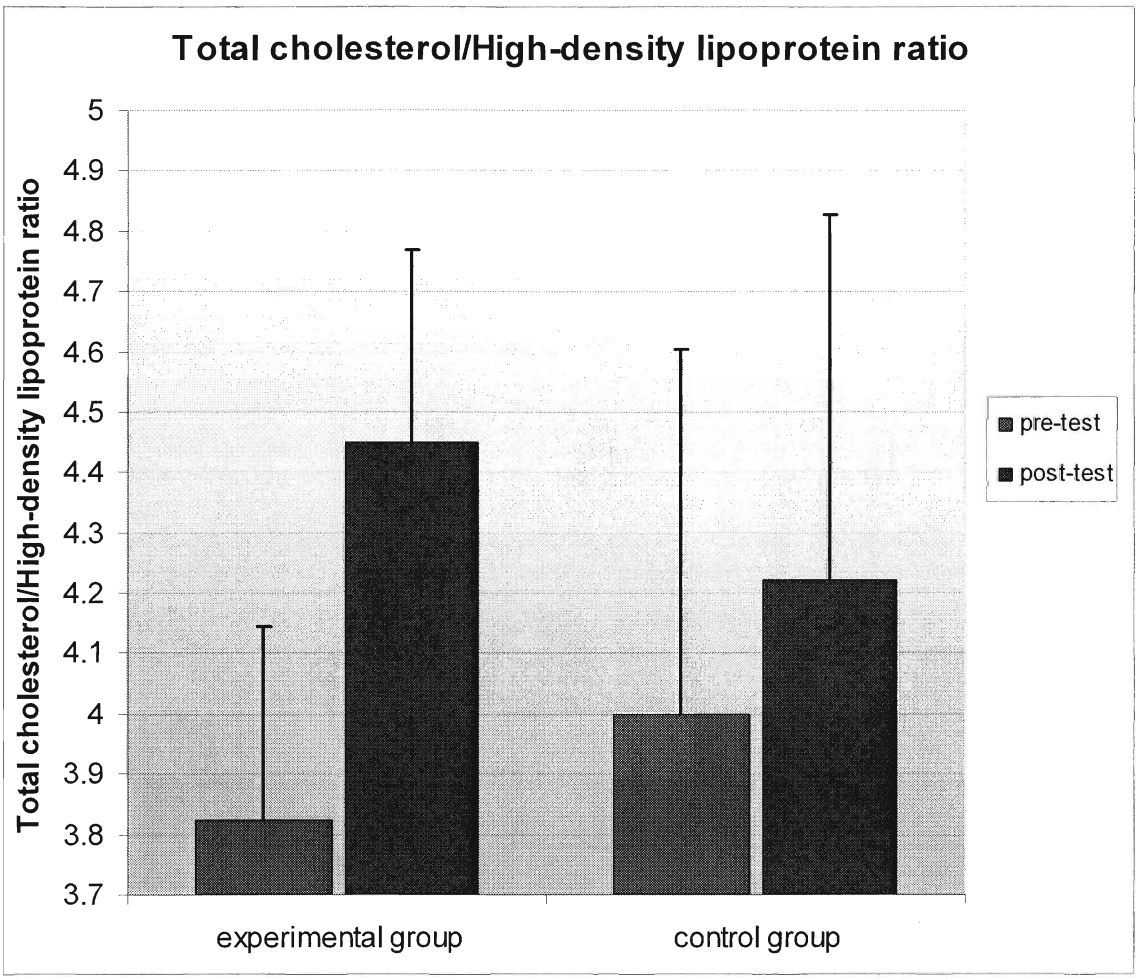
Values are means ± SEM. Asterisk shows statistically significant change ($p < 0.05$).

Figure 12. Values for total cholesterol for control and experimental groups (pre- and post-tests).



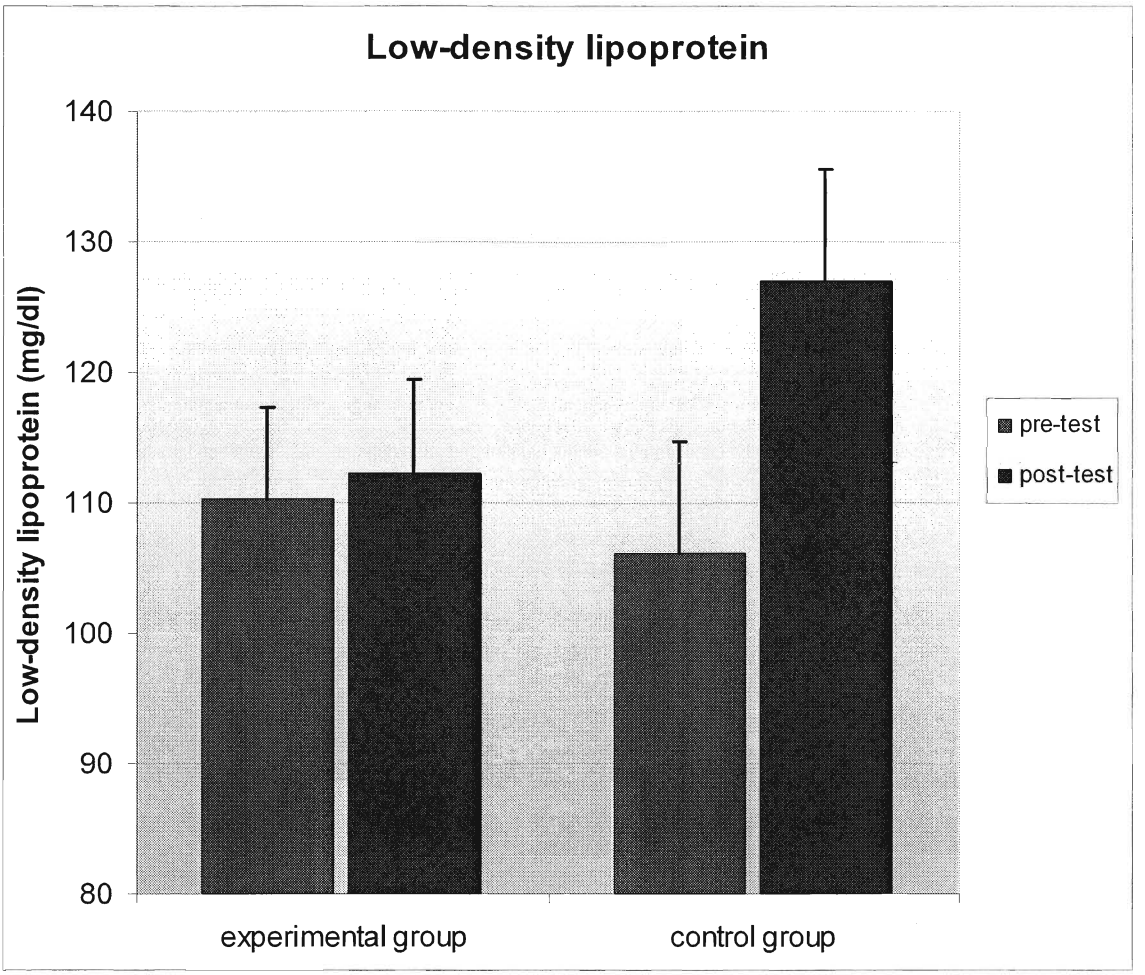
Values are means ± SEM. Asterisk shows statistically significant change ($p < 0.05$).

Figure 13. TC/HDL cholesterol for control and experimental groups (pre- and post- tests)



Values are means ± SEM. Asterisk shows statistically significant change ($p < 0.05$).

Figure 14. LDL in experimental and control groups (pre – and post-test)



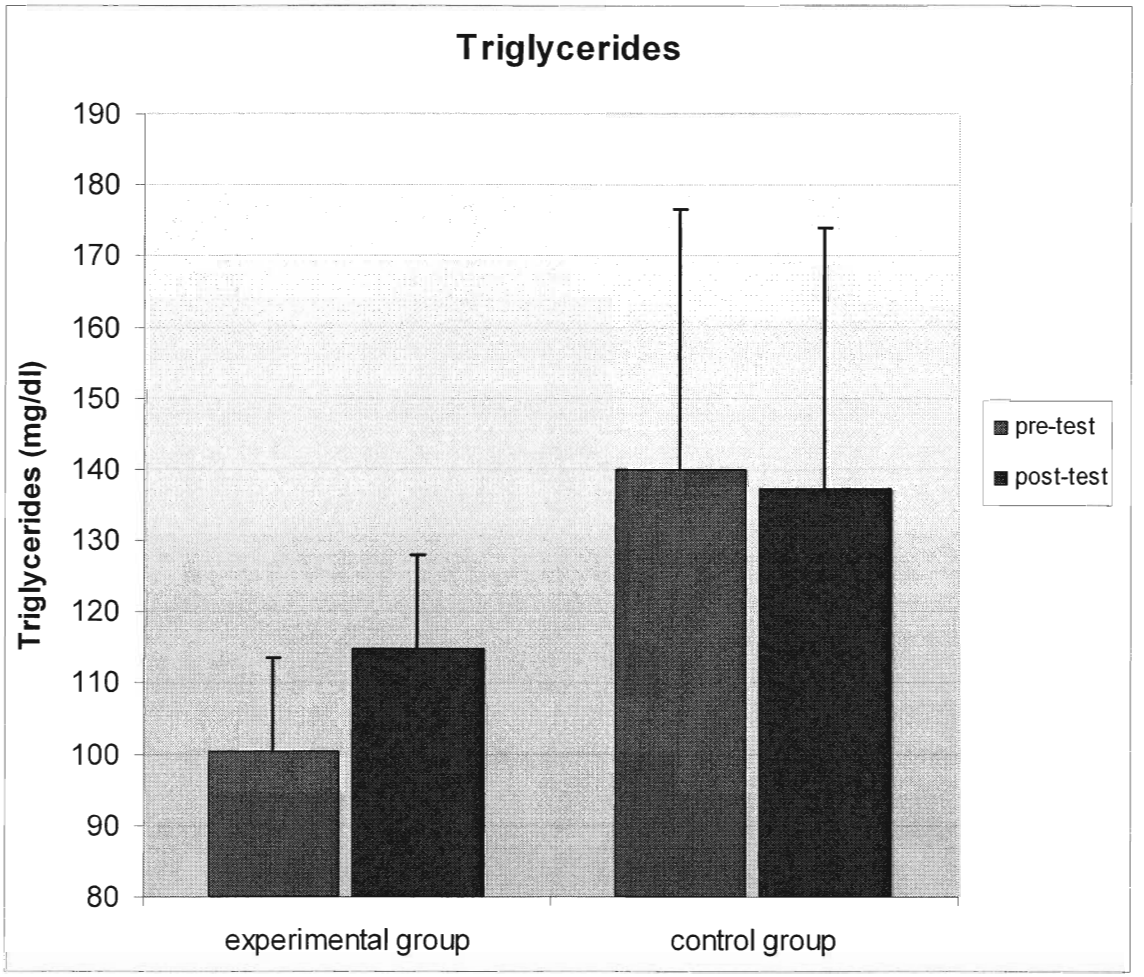
Values are means ± SEM. Asterisk shows statistically significant change ($p < 0.05$).

In terms of blood triglycerides, neither experimental nor control group exhibited any change. For example, pre- and post-values for experimental and control groups were 100.3 $\pm$ 19.6 versus 114.8 $\pm$ 15.3 and 140 $\pm$ 23.5 versus 137.3 $\pm$ 17.9 mg/dl (Fig 15). Percentage changes for HDL, TC, TC/HDL, LDL and TRG are summarized in Fig.17.

4.4 Blood Glucose

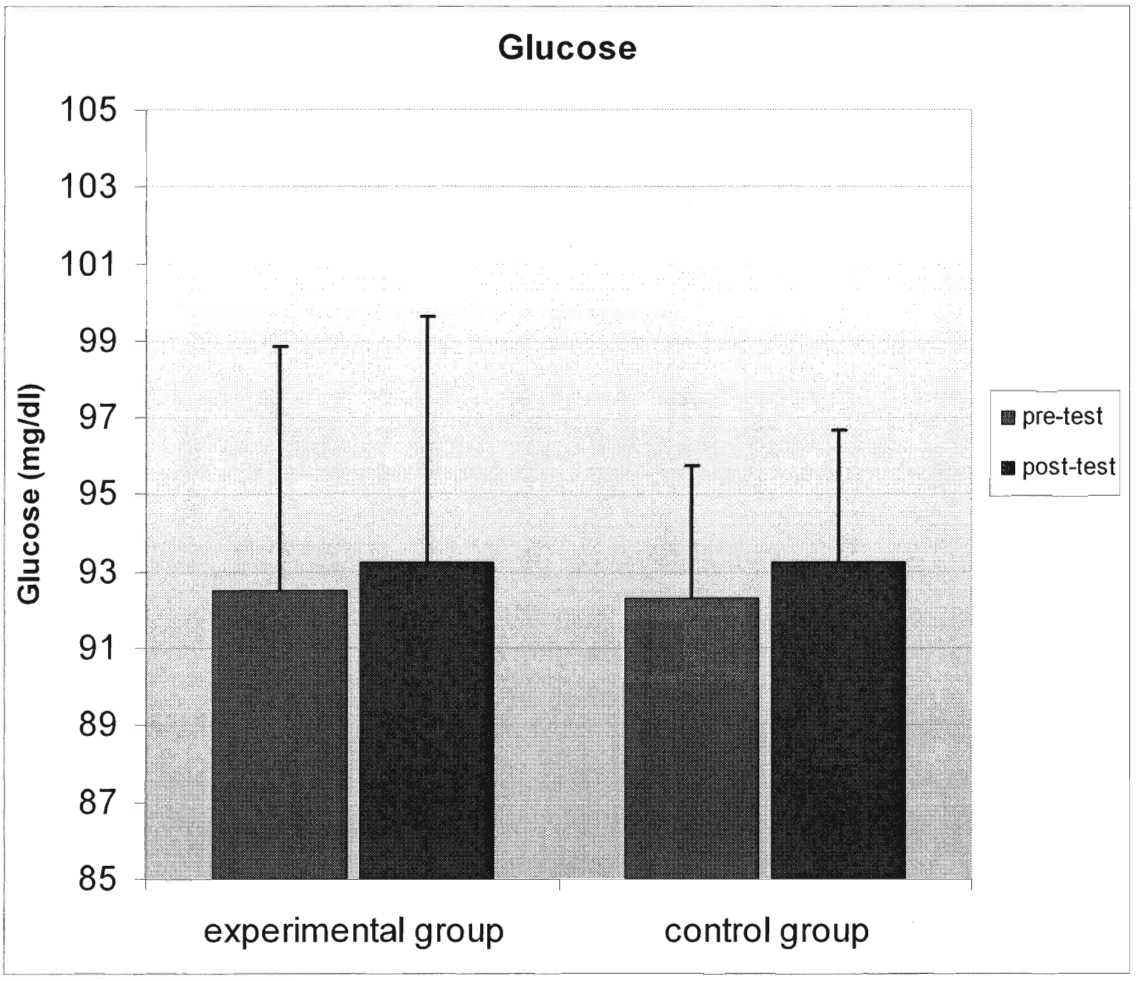
Resting blood glucose levels for experimental and control groups are shown in Fig. 16. These data reveal that pre- and post values for both experimental and control groups were unchanged. As an example, pre- and post-values for experimental and control groups were 92.5 $\pm$ 4.8 versus 93.3 $\pm$ 4.3 mg/dl and 92.3 $\pm$ 11.3 versus 93.2 $\pm$ 2.6 mg/dl, respectively. Percentage changes for blood glucose are also shown in Fig.17.

Figure 15. Blood triglycerides for experimental and control groups (pre- and post-tests)



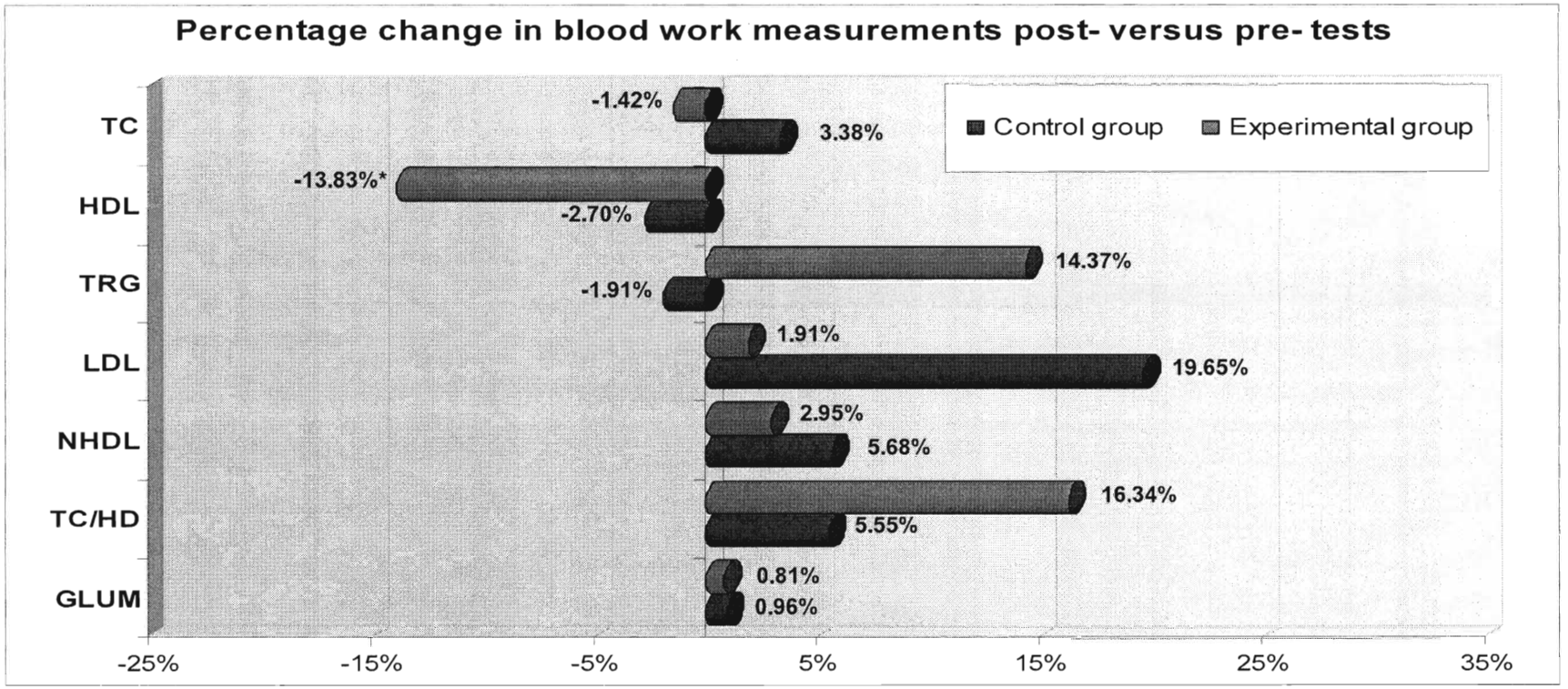
Values are means ± SEM. Asterisk shows statistically significant change ($p < 0.05$).

Figure 16. Blood glucose for control and experimental group (pre- and post- tests)



Values are means ± SEM. Asterisk shows statistically significant change ($p < 0.05$).

Figure 17. Percentage change in blood lipids and blood glucose for control and experimental groups (pre-and post-test).



Values are means ± SEM. Asterisk shows statistically significant change ($p < 0.05$). % change calculated as post-pre/pre x 100. HDL levels of the experimental group decreased by 13.8 ± 1.2% while that of the control group did not change. On the other hand, pre- and post-values for TRG, LDL, NHDL, TC/HDL and GLU did not change ($P < 0.05$).

4.5 Blood Pressure

For experimental group pre-test mean value was 131 ± 2 mm/Hg, post-test value 129 ± 2 mm/Hg. For control group pre-test mean value was 123 ± 2 mm/Hg, post-test value 125 ± 2 mm/Hg.

For experimental group pre-test mean value was 84 + 2 mm/Hg, post-test value 83+ 2 mm/Hg. For control group pre-test mean value was 81 + 1 mm/Hg, post-test value 82 + 1 mm/Hg.

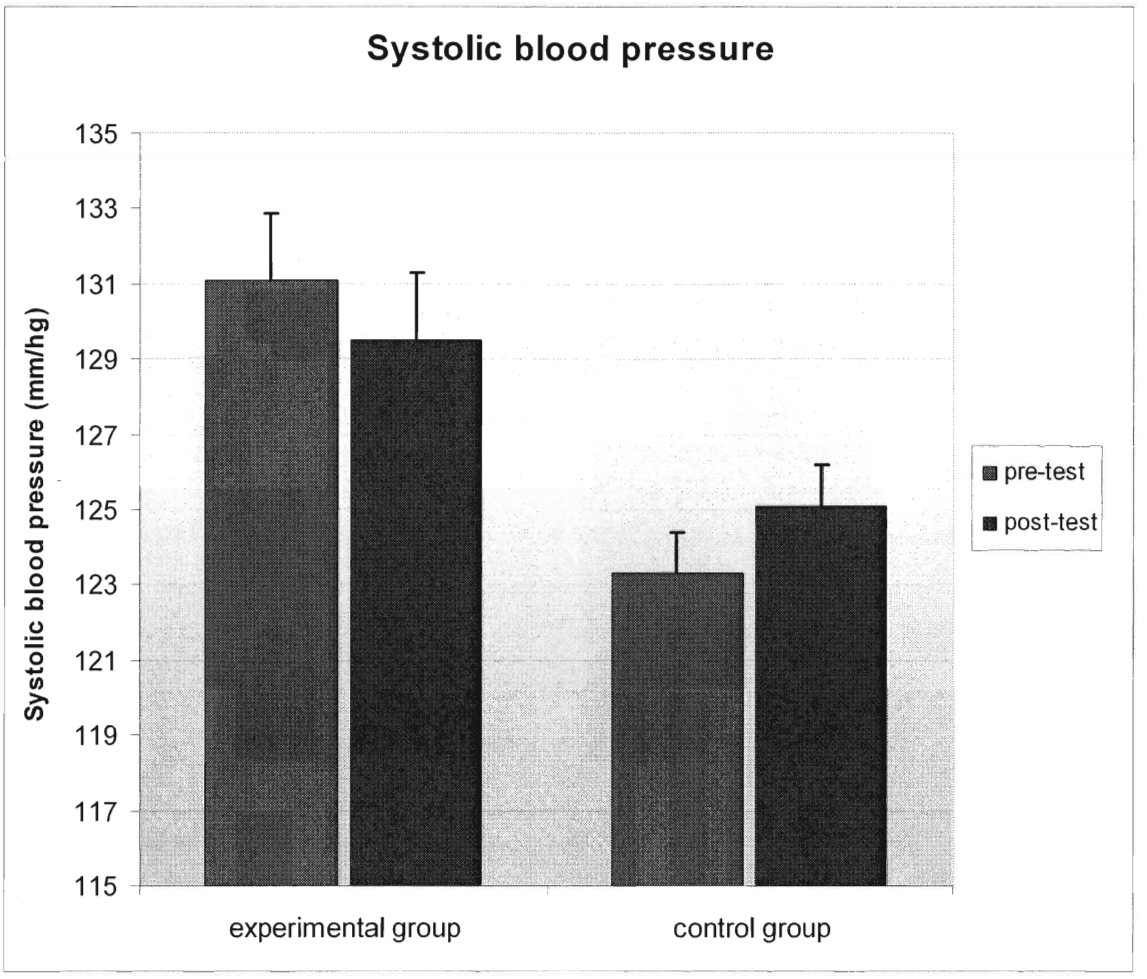
For experimental group pre-test mean value was 47± 1 mm/Hg, post-test value 47 ± 2 mm/Hg. For control group pre-test mean value was 42 ± 2 mm/Hg, post-test value 43 ± 1 mm/Hg.

For experimental group pre-test mean value was 127± 2 mm/Hg, post-test value 125 ± 2 mm/Hg. For control group pre-test mean value was 122 ± 2 mm/Hg, post-test value 123 ± 2 mm/Hg

4.6 Framingham Point Score

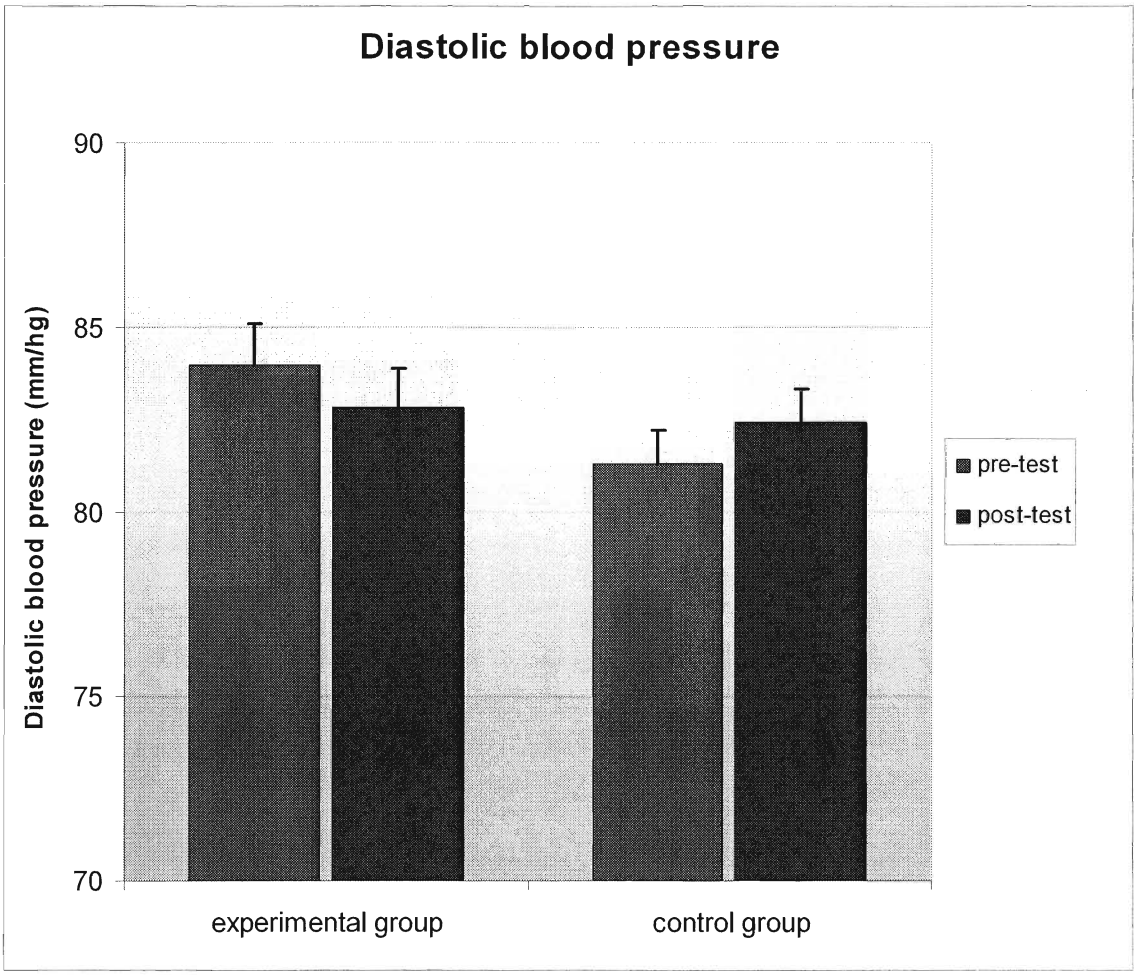
For experimental group pre-test mean value was 1.4± 0.9 points, post-test value 2.7 ± 0.7 points. This represents statistically significant change. For control group pre-test mean value was 1.8 ± 1 points, post-test value 1.8 ± 0.9 points.

Figure 18 Systolic blood pressure for control and experimental groups (pre- and post-test)



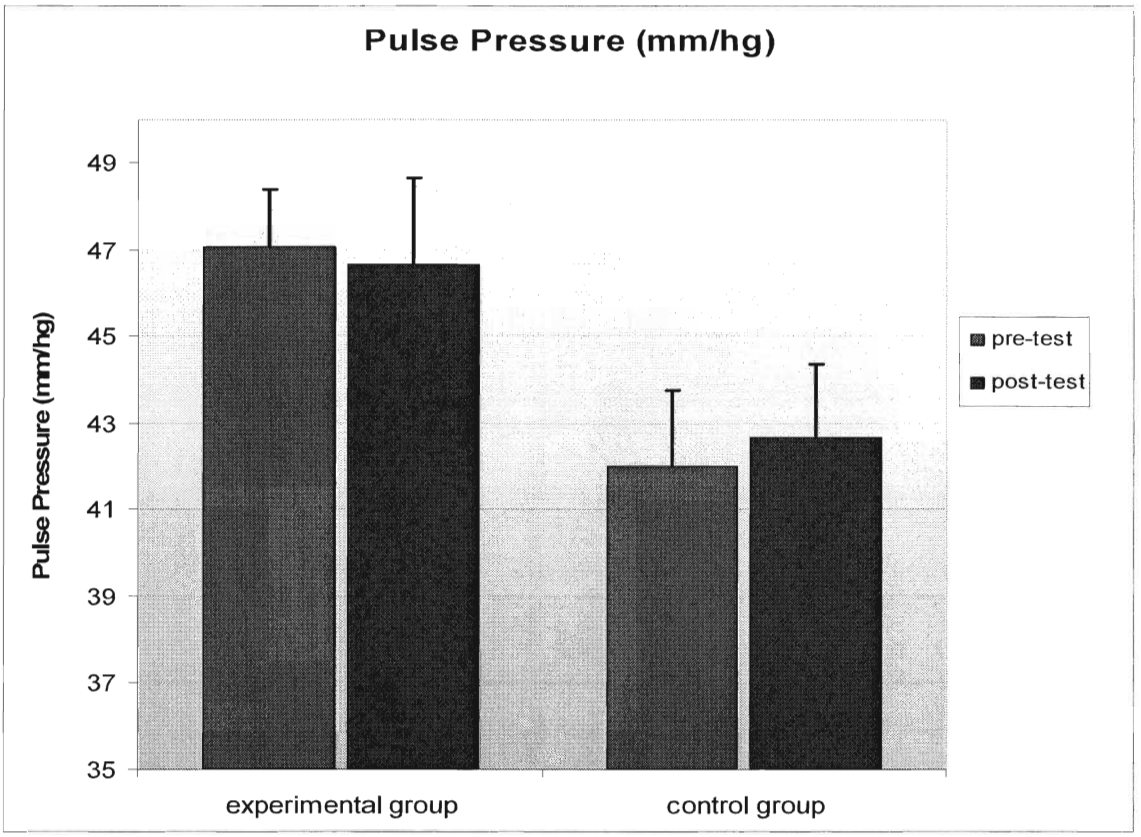
Values are means ± SEM. Asterisk shows statistically significant change ($p < 0.05$).

Figure 19. Diastolic blood pressure for control and experimental group (pre- and post-test)



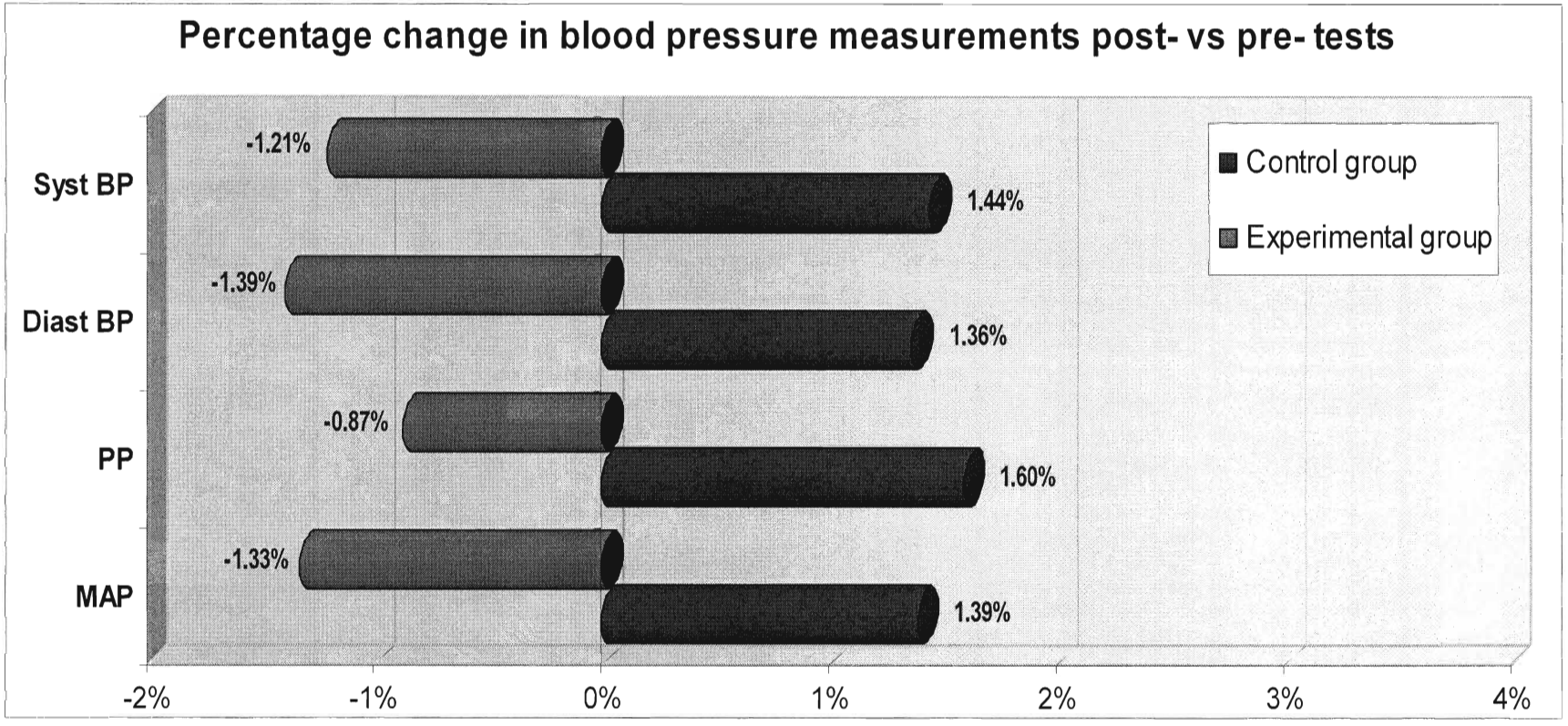
Values are means ± SEM. Asterisk shows statistically significant change ($p < 0.05$).

Figure 20. Pulse pressure for control and experimental group (pre- and post- test)



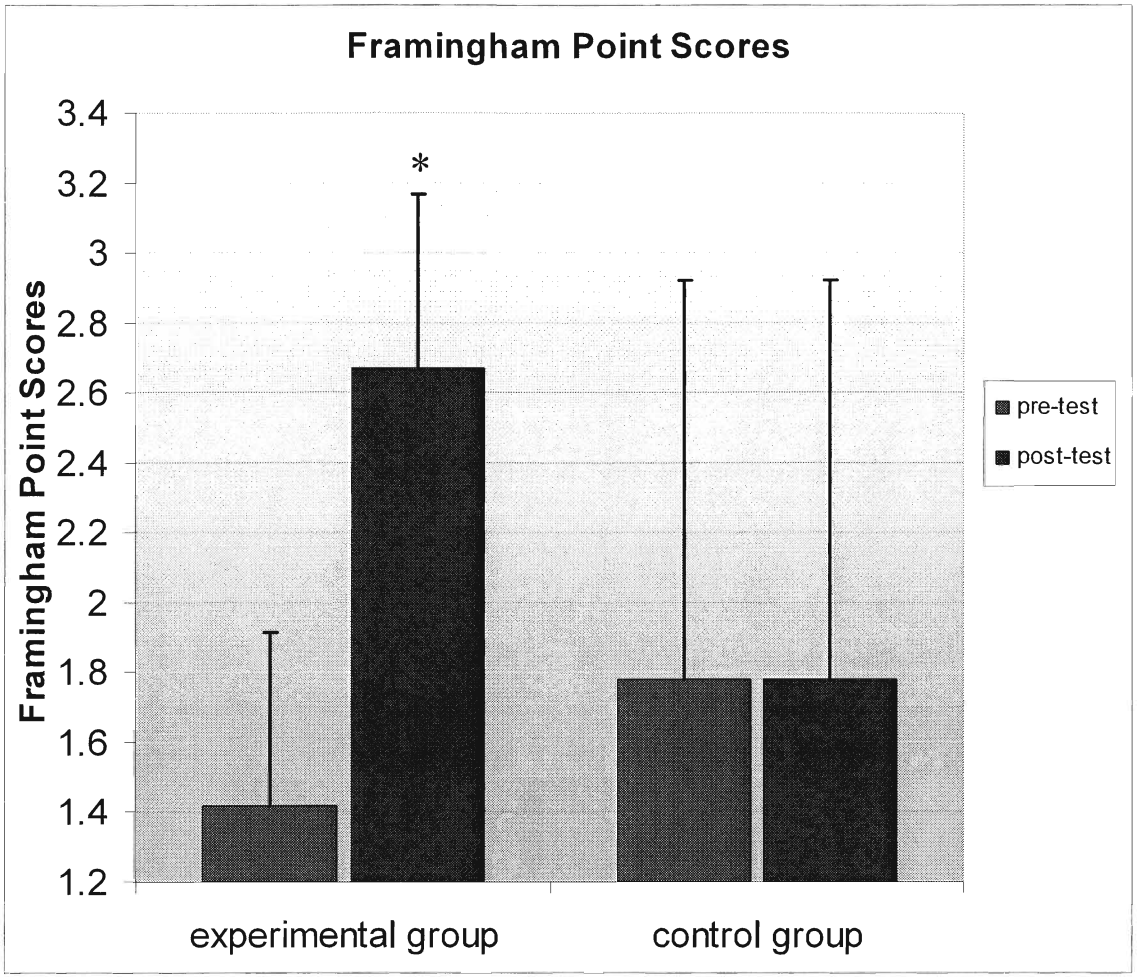
Values are means ± SEM. Asterisk shows statistically significant change ($p < 0.05$).

Figure 21. Blood pressure parameters in control and experimental group (pre- and post test).



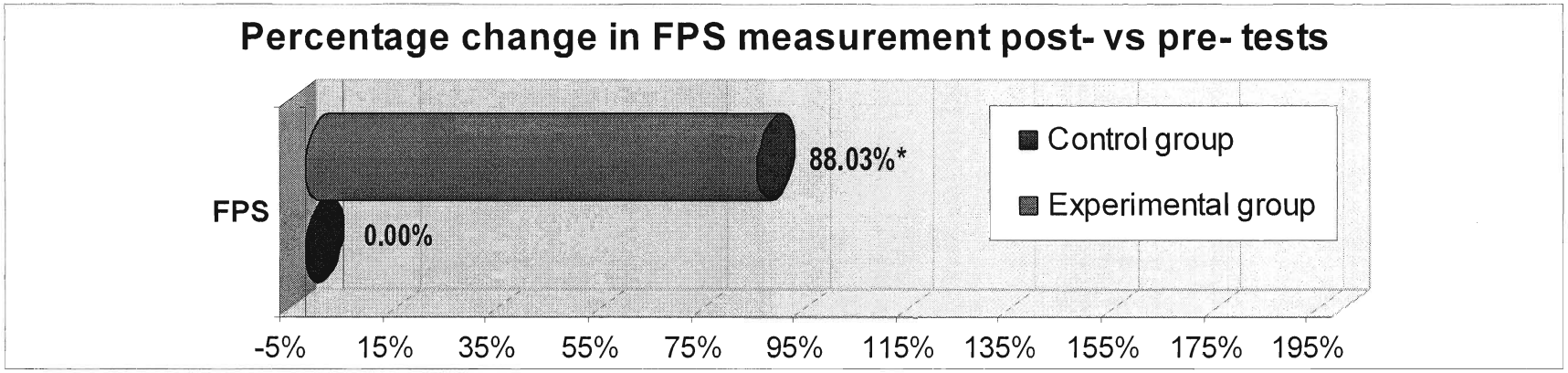
Values are means ± SEM. Asterisk shows statistically significant change ($p < 0.05$). % change calculated as post-pre/pre x 100. No blood pressure parameter was altered in either experimental or control group.

Figure 22. Mean values of Framingham point score for control and experimental group (pre- and post- test).



Values are means ± SEM. Asterisk shows statistically significant change ($p < 0.05$).

Figure 23. FPS for CAD risk in experimental and control groups (pre- and post-tests)



Values are means ± SEM. Asterisk shows statistically significant change ($p < 0.05$). FPS increased by 88.03% in the experimental group. FPS did not change in the control group.

CHAPTER 5: DISCUSSION

Our study examined the effects of interval training in a form of ball hockey on coronary risk factors in a population of sedentary, middle aged men. The rationale for this approach was recent work showing that IT provides a powerful stimulus for improvement in aerobic and anaerobic indices of fitness (109). A motivation for this study was the expectation that participation in high intensity recreational sport, in this case ball hockey, would prove to be a viable alternative for training to improve outlook regarding CVD. Although total training time represented by ball hockey participation falls well below the minimal threshold required for fitness improvements in many studies, it was hoped that the highly intense nature of this exercise would provide a useful adjunct to more standard training / exercise programs (1,4).

5.1 Epidemiology of exercise and CVD

Blair et al. (30) reported that only a modest level of fitness is necessary to achieve the benefits of reduced mortality from CVD, in persons without coronary artery disease. Pollock et al. (130) exercised six groups of healthy males in order to improve cardio respiratory fitness. Intensity was the same for all groups. Three groups exercised for 15, 30 and 45 minutes per session for 3 days a week while the other three groups exercised at varying frequencies one, three and five times per week for thirty minutes per session. The conclusion was that the improvements in fitness were related to the overload. In our study we did not use the rate of perceived exertion to quantify intensity of the players activity. However, the data that we obtained show that exercise was able to prevent weight gain in

the experimental group but were not more beneficial for the lipoprotein profile than was a sedentary lifestyle.

5.1.1 Effects of exercise on body composition

Furthermore, in regards to body composition, our data for experimental group has shown positive trend for Lean Body Mass, Body Mass Index as well as for Body Fat Percentage, which had statistically significant change. Due to importance of waist-to-hip ratio, it is important to mention that experimental group has shown positive trend in that variable as well. The waist-to-hip ratio is a highly significant predictor for development of cardiovascular disease (171). That means that, each increase in the waist-to-hip ratio is associated with an increase in the risk of a heart attack (171). Apparently, this is because of the type of fat that builds up within the abdomen, which causes a large waist size and has greater ill health effects than fat located in other places (167). These fat cells in the belly do more damage because they release fatty acids directly to the liver. Having said that, health implications of waist –to- hip ratio are significant because it increases the estimate of cardiovascular disease attributable to obesity (171). Although the effect of exercise on visceral adiposity per se was not measured in our study, decreased body fat percentage in the experimental group suggests decreased obesity.

5.1.2 Cardiorespiratory endurance as a indicator of health

Epidemiological data have established that physical inactivity increases the incidence of unhealthy conditions that results in premature death (1). It is known that elevated levels of LDL combined with low levels of HDL are strongly related to the development of coronary atherosclerosis (98). Wanamathe (160) examined the relationship between

physical exercise and the incidence of CVD and two important findings emerged: first, an inverse association between physical exercise and the incidence of CVD, and secondly, the relative risk of a sedentary lifestyle appeared to be similar in magnitude to that of other coronary risk factors. The results obtained were approximately similar to those that provided the benefit in preventing cardiovascular events and death from any cause among subjects in the Harvard Alumni Study (143). In particular, the data from our ball hockey study reveal that exercise performed twice per week in duration of 30 minutes improves cardio respiratory endurance (CRE), in the form of VO2 Max in a previously sedentary population. VO2 Max was our index of CRE. Cardio respiratory endurance represents ability to perform dynamic exercise using large muscle groups at moderate to high intensity for prolonged periods (5). CRE is a good indicator of overall health. Low levels of cardiorespiratory endurance from a sedentary lifestyle have been associated with an increased risk of premature death from cardiovascular disease (143). Moderate to high levels of prolonged physical activity can improve cardiorespiratory endurance (5).

5.1.3 VO2 Max as a index of CRE

What is currently known is that VO2 Max increases substantially following endurance training (109,116), but that the amount of increase is limited in each individual. It is generally accepted that VO2 Max as the best indicator of cardiorespiratory fitness (4,116 168). Exercise in duration of 20 minutes per session at a minimum intensity of 60% maximal heart rate, performed 3 times weekly improves VO2 Max (115). An increase of 15 to 20 percent is typical for an average person who was sedentary before training and who trains at 75% of their capacity three times per week (115). The VO2 Max of a sedentary individual can increase from an initial value of 35 ml-kg-min to 42 ml-kg-min

as a result of such a program. Lemura et al (115) carried out a Meta analysis of the effect of such training on VO2 Max. 25 of 27 studies showed significant improvement in VO2 Max. They found that those improvements were greater when intensity was at 80% of maximal heart rate. They also noted that durations of exercise in length of 30 minutes produced greater improvement compared to durations less than this time. The major-limiting factor for VO2 Max increase appears to be oxygen delivery to the active muscles (155).

5.1.4 Exercise and blood pressure

Our study warrants additional conclusions. First, no change in the overall blood pressure profile was observed in our study. It is clear that aerobic exercise training can reduce the resting blood pressure among sedentary, normotensive and hypertensive adults (163,109,104). Higher levels of physical activity and greater fitness levels are associated with reduced incidence of hypertension (163). A meta analysis of the literature conducted by Miller et al. (123) revealed an average reduction of 6 to 9 mm/Hg for systolic and diastolic blood pressure in patients with mild to moderate hypertension. However, many of the studies of exercise on hypertension had small numbers of participants. Cooper et al. (56) investigated the effect of a six-week programme of moderate intensity exercise on blood pressure among unmediated, sedentary adults aged 25-63 years with 150 mmHg to 180 mmHg systolic and/or 91 mmHg to 110 mmHg diastolic blood pressure. The control group performed 30 minutes of moderate intensity exercise (brisk walking) five days per week for six weeks and was compared with control group, which maintained existing level of physical activity. The reduction in mean blood pressure between baseline and six-week follow-up was greater in the intervention group than in the control group for

both systolic and diastolic blood pressure but the net hypertensive effect was not statistically significant. The conclusion of this study was that in order to achieve a statistically significant magnitude of the hypotensive effect higher level exercise intensity was required (56). It was also noted that expectation of general practitioners and patients that a programme of moderate intensity exercise will lead to a clinically important reduction in the individual's blood pressure was unlikely to be achieved. Hulbert et al (83) tried to identify the features of an optimal exercise programme in terms of type of exercise, intensity and frequency that would maximize the training induced decrease in blood pressure. The inclusion criteria for this Meta analysis study were limited to randomized controlled trials of aerobic exercise conducted over a minimum of 4 weeks where systolic and diastolic blood pressure was measured. A total of 26 studies used aerobic exercise training. Aerobic exercise training reduced systolic blood pressure by 4.7 mm Hg and diastolic blood pressure by 3.1 mm Hg as compared to a non-exercising control group. The blood pressure reduction seen with aerobic exercise training was independent of the intensity of exercise and the number of exercise sessions per week. The conclusion of this study was that aerobic exercise training had a small but significant effect in reducing systolic and diastolic blood pressure. The author also stated that increase of exercise intensity above 70% VO2 Max or increase of exercise frequency to more than three sessions per week did not have any additional impact on reduction of blood pressure.

5.1.5 Exercise and HDL profile

In our study we noted that, although the two groups in our study had similar lipoprotein profile, the exercise group had a statistically significant decrease in the HDL profile. One of the factors that might influence reduction of HDL in our study could be poor nutrition of our participants. That factor could not be controlled by our side and according to several studies (77,100, 102,166, 170, 172) poor nutrition can damage HDL at which point it no longer does any of good things and instead actually contribute to cardiovascular disease by winding up with LDL in plaque. There are three main reasons this happens.

1) The failure to provide adequate nutrition to re-energize HDL after it has been out working. This leads to a lack of apoA-I and an HDL cell membrane that has lost functionality, which can lead to reduction in HDL (77).

2) Oxidative damage to apoA-I, caused by inflamed and overheated immune cells. This means individuals with inflammatory health issues will have poor quality HDL. The greater the inflammation the worse the HDL quality and quantity (77).

3) Sugar glycation of HDL. The more uncontrolled the blood's sugar level, the worse the HDL problem (170).

According to our study, it appears that adverse effect of amount of exercise alone on HDL levels is not as large as originally thought. Exercise may improve the HDL cholesterol levels particularly among participants with lower BMIs. From the analysis from several studies (170,99,100), larger changes were seen in subjects who also lost

weight. Zmuda and associates (172) concluded that exercise training has little effect on HDL levels and metabolism in men with initially low HDL cholesterol. This study compared the HDL response to 12 months of endurance exercise training without weight loss in 17 men aged 26-49 years with initially low HDL levels. They concluded that the ability to increase HDL levels through endurance exercise in subjects with low initial HDL is limited because exercise fails to alter triglyceride. Apparently, the response of HDL levels will differ for each individual depending on the intensity, duration and frequency of exercise, the initial HDL level, and the length of the training period (3). Williams et al. (166) have concluded that HDL can be improved by exercise, but a volume of activity must be in between 1000 and 1500 calories utilized per week, for six months or more. It seems that there is an exercise threshold for exercise intensity, amount of exercise, and length of the training period, that must be met before changes in HDL are evident (3) but the precise level has yet to be elucidated (39). Kodama et al. (102) conducted a meta-analysis examining the effect of exercise on HDL levels. On average, participants in these studies exercised for 40 minutes, 3 to 4 times per week, and the effect on HDL was measured after 8 to 27 weeks. Across the studies, participants had modest increase of HDL, averaging about 2.5 mg/dl. The most interesting finding is the observation that the duration of exercise session correlated the best with rises in HDL levels. From this meta-analysis study, it can be concluded that exercise of 3 to 4 times a week with sessions between 20 and 40 minutes can lead to increase of HDL levels. In fact, increasing the duration of exercise session appears to be the best way to increase level of HDL. Altogether, our findings and previous studies suggest that the relationship

between HDL levels and exercise are complex and relate to several variables, such as exercise duration, exercise intensity and lipid profiles of the participants

5.2 Ball Hockey as a model for Interval Exercise

Recreational ball hockey is a sport widely played in Canada and USA (over 100,000 players). The game format is very similar to organized ice hockey. Each team consists of 10 - 12 individuals with players taking turns or "shifts" on the floor. Games consist of three 10 minutes periods incorporating stop time. While on the floor, each player is required to perform repeated sprints, with short rest period in between. Longer periods of recovery are experienced while players return to the player's bench between shifts. In general, ball hockey play typically involves repeated periods of high-intensity work performed for short durations (5 -10 s) mixed with periods of lower-intensity work such as jogging, walking or standing. In the scientific literature sports eliciting these characteristics have been termed 'intermittent' (5, 20). Moreover, ball hockey play requires an extensive amount of eccentric muscle contractions while engaged in turning, stopping and/or reversing direction as well as a considerable amount of upper body work (shooting, passing, checking etc). Thus, based on the above, even though the actual work is difficult to quantify, ball hockey may represent an appropriate surrogate for IT models. It is well established that intensive interval training stresses recruitment and adaptation of type 2(fast twitch) muscle fibers that are remarkably and equally responsive as type 1 muscle fibers (slow twitch) in their ability to increase mitochondrial enzyme activity to high absolute levels (55). Intense interval work utilizes a greater percent of the body's

muscles, both slow and fast twitch. It also places added energy demands on the respiratory system, cardiovascular system and nervous system (59). This is the reason why more fat and glycogen are burned to support the expanding energy demands of the body during - and after - intense exercise (156). That means that the cost of short intense interval exercise is very high in terms of energy demands in comparison to low intensity aerobic exercise. What's more, while at rest trained active muscles burn more fat night and day, contributing to further fat loss (51,69). Tremblay and Bouchard (156) analyzed large sample size of data with 1257 male subjects, which proved that those involved in IT have less subcutaneous fat than those not involved. In 1994 (34) the same authors completed another study, which concluded that IT is the best for fat loss. In this study they divided subjects in endurance training group for 20 weeks and IT group for 15 weeks. They have found that IT creates a bigger loss of subcutaneous fat. Data suggest that high intensity interval training can induce changes in mitochondrial potential and still stimulate performance aside from changes in mitochondrial potential. For example, Le Blanc (113) has shown that 5-8 weeks of SIT increases skeletal muscle blood flow while Ortebland (128) has found increase in sarcoplasmic reticular function. Jacobs (95) has found increased lactate transport capacity and H release from active muscle. The most conclusive evidence so far came from Burgomaster et al. (45). These authors hypothesized that sprint interval training (SIT), or repeated sessions of high-intensity exercise, would induce rapid changes in transport proteins associated with CHO metabolism, whereas changes in skeletal muscle fatty acid transporters would occur more slowly. They concluded that short-term SIT induces rapid increases in skeletal muscle oxidative capacity but has divergent effects on proteins associated with glucose, lactate,

and fatty acid transport. In their study they confirmed the findings of Barnet (23) that SIT induces changes in monocarboxilate transporter protein, which is responsible for regulation of lactate and H exchange in skeletal muscle. Burgmaster et al. (45) also found an increased GLUT4 protein content which theoretically could facilitate higher glucose uptake during recovery and explain in part the higher muscle glycogen content observed after SIT. This finding confirms earlier findings (44) to suggest that SIT type training is a powerful stimulus for improving carbohydrate metabolism and both aerobic and anaerobic energy profiles of trained muscles.

There is no scientific data available concerning the time and motion characteristics of ball hockey. However, time-motion analysis conducted at the 2007 World Championship by the Canadian Ball Hockey Association revealed the following: average player shift duration was ~ 45 seconds and during this interval players covered a distance of ~ 200 meters. There is, however, no existing physiological data concerning the physiological responses to or the adaptations to chronic ball hockey play. Indeed, our data is the first scientific examination of the physiological adaptation to this growing sport.

5.3 Study Limitations

Certain limitations must be considered when evaluating the overall significance of this study. These limitations span the range from measurement error to subject age, fitness level and compliance.

In our study, The Rockport Fitness Walking Test, a maximal paced 1-mile walk test was used to evaluate cardiorespiratory endurance through the estimation or prediction of

maximal oxygen consumption (VO_2max) (110). Since the original validation study by Kline, et al. (110), the Rockport Test has been cross validated in many samples (110, 119, 159,162), all of which have supported the accuracy of this field test. In the study of Kline et al (110), reliability coefficients (test-retest) associated with the original maximally paced Rockport Test were reported to be .93 for heart rate and .98 for walking time (1). As a result of this and some other studies (119, 159, 162), the Rockport Walking Test is considered a valid and reliable field test to assess the aerobic performance, because walking technique and the use of a steady-state walking pace does not adversely influence the reliability of estimating maximal oxygen consumption. However, standardized laboratory tests for determination of Vo2 Max is more accurate since instruments are more precise and motivation of participant is a factor in order to determine true Vo2 Max.

The training effect we found for our experimental subjects (11% increase in VO2 Max) may not be elicited in younger subjects with higher levels of pre-existing fitness.

We did not quantify the training load, either in terms of exercise heart rate or time and motion analysis, represented by ball hockey game participation.

We did not use Rate of Perceived Exertion to monitor intensity of the games.

The HDL cholesterol measurements reported for the experimental group were inconsistent with what is normally reported for interval training type interventions (62,86,102.103). Indeed, the fact that HDL levels consistently decreased for subjects in both the experimental and control groups may be considered an anomalous result due to systematic measurement errors related to equipment calibration. This contention is

supported by the fact that post-test results were consistently lower than pre-test results on a subject by subject basis (see appendix D).

Related to the above, subject lifestyle was not monitored or scrutinized. It is thus possible that seasonal changes in diet contributed to anomalous HDL results.

The intervention may have been too brief or too sporadic to produce consistent reductions in various coronary risk factors. For example, the number of games played (maximum of 16) by the experimental group subjects may have been insufficient of an exercise intervention to reduce blood pressure or blood lipids. In this case, it might be expected that prolonged participation in recreational ball hockey would provide a more powerful stimulus to reduce coronary risk factors.

The results of this study are limited to sedentary males aged 30 - 60 and may not apply to younger participants of either sex. The results of this study are limited to one season of ball hockey participation and may not apply to other longer periods of participation or to other sports.

We did not evaluate other aspects of fitness such as anaerobic power or muscle strength. Thus, our study was insensitive potential improvements in these fitness parameters.

The use of the Framingham Point Score (FPS) or similar methods that integrate individual risk factors to derive an overall "coronary risk" may in of itself be considered a limitation. Because the FPS "bins" individual scores, relatively large changes (positive or negative) in the middle of a "bin range" may not be reflected in the overall score. On

the other hand, small changes (positive or negative) near the extreme ends of a "bin range" may actually be reflected in the overall score.

Blood lipid and blood glucose measurements were dependent on subject compliance in regard to prior fasting.

Body composition measurements (e.g. % body fat) were dependent on subject compliance regarding prior water intake.

Subject activity levels during the study were not closely monitored. Thus, we cannot exclude the possibility that improvements in cardiorespiratory fitness (i.e. VO2 Max) reported determined for the experiment group was due to non ball hockey related activities.

5.4 Conclusions

This study was the first to study the effect of recreational/competitive ball hockey on cardiovascular risk factors in previously sedentary adult males. By extension, this study has provided some insights into the effectiveness of organized sport (recreational) to reduce cardiovascular risk factors in middle-aged men. The results of our study demonstrate that even a small amount of intense interval training is somewhat efficient in reversing the adverse impact of some CRFs. The closely supervised ball hockey regimen is a feature of our study and demonstrates the importance of such training in reducing some CRF. Exercise training, especially interval, appears to be health beneficial to the sedentary population. Although larger prospective studies that will use ball hockey as an intervention are needed to advance our conclusions, we believe that ball hockey yield

very favorable results in regards to reduction of some cardio vascular risk factors by increasing VO2 Max while reducing body fat percentage. It is also noteworthy to mention that recreational sport may be useful for adding intensity component to overall fitness training. The present study suggests that exercise in the form of ball hockey is partially able to reduce coronary risk factors, suggesting that this may be a promising treatment strategy for cardio vascular disease. Given the positive (but non-statistically significant) trends in our blood pressure, blood lipid and anthropometric data, further studies employing either more subjects, higher frequency of games and/or more games, are warranted. More powerful scientific models incorporating the above features may find statistically significant changes with bigger effect size for all variables.

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APPENDIX A - SUMMARY STATISTICS FOR CONTROL GROUP

		Mean	N	Std. Deviation	Std. Error
Pair 1	Syst BP	123.33	9	6.000	2.000
	Syst BP 2	125.11	9	6.373	2.124
Pair 2	Diast BP	81.33	9	3.240	1.080
	Diast BP 2	82.44	9	4.003	1.334
Pair 3	PP	42.00	9	5.339	1.780
	PP 2	42.67	9	5.074	1.691
Pair 4	MAP	122.0333	9	4.51225	1.50408
	MAP 2	123.7311	9	5.53567	1.84522
Pair 5	wh ratio	.929671	9	.0248530	.0082843
	wh ratio 2	.932898	9	.0286146	.0095382
Pair 6	bodyfat	22.722	9	4.0993	1.3664
	bodyfat 2	22.167	9	3.8945	1.2982
Pair 7	BMI	26.256	9	2.1518	.7173
	BMI 2	26.222	9	1.9531	.6510
Pair 8	tc	190.67	9	36.776	12.259
	tc 2	197.11	9	48.339	16.113
Pair 9	hdl	49.67	9	10.920	3.640
	hdl 2	48.33	9	12.430	4.14
Pair 10	trg	140.00	9	70.551	23.517
	trg 2	137.33	9	53.659	17.886
Pair 11	ldl	106.14	7	29.785	11.258
	ldl 2	127.00	7	39.846	15.060
Pair 12	nhdl	140.89	9	36.798	12.266
	nhdl 2	148.89	9	44.675	14.892
Pair 13	tc hdl	4.000	9	1.1715	.3905
	tc hdl 2	4.222	9	1.0616	.3539
Pair 14	glumg	92.33	9	11.258	3.753
	glumg 2	93.22	9	7.710	2.570
Pair 15	HR bpm	118.56	9	18.855	6.285
	HR bpm 2	122.33	9	17.909	5.970
Pair 16	time mins	15.0511	9	1.52194	.50731
	time mins 2	15.0478	9	1.44957	.48319
Pair 17	VO2 Max	40.56	9	3.087	1.029
	VO2 Max 2	40.33	9	3.041	1.014
Pair 18	ECW TBW	.37478	9	.005869	.001956
	ECW TBW 2	.37511	9	.005578	.001859
Pair 19	BMR	1786.56	9	163.847	54.616
	BMR 2	1794.78	9	153.139	51.046
Pair 20	FSP	1.78	9	3.073	1.024
	FSP 2	1.78	9	2.819	.940

APPENDIX B - PAIRED SAMPLES CORRELATIONS FOR CONTROL GROUP

		N	Correlation	Sig.
Pair 1	Syst BP & Syst BP_2	9	.865	.003
Pair 2	Diast BP & Diast BP_2	9	.739	.023
Pair 3	PP & PP_2	9	.614	.079
Pair 4	MAP & MAP_2	9	.872	.002
Pair 5	wh ratio & wh ratio_2	9	.343	.366
Pair 6	bodyfat & bodyfat_2	9	.359	.343
Pair 7	BMI & BMI_2	9	.600	.088
Pair 8	tc & tc_2	9	.682	.043
Pair 9	hdl & hdl_2	9	.471	.201
Pair 10	trg & trg_2	9	-.556	.120
Pair 11	ldl & ldl_2	7	.825	.022
Pair 12	nhdl & nhdl_2	9	.382	.311
Pair 13	tc_hdl & tc_hdl_2	9	-.325	.394
Pair 14	glumg & glumg_2	9	.463	.210
Pair 15	HR bpm & HR bpm_2	9	.457	.216
Pair 16	time_mins & time_mins_2	9	.770	.015
Pair 17	VO2 Max & VO2 Max_2	9	.470	.201
Pair 18	ECW_TBW & ECW_TBW_2	9	.933	.000
Pair 19	BMR & BMR_2	9	.990	.000
Pair 20	FSP & FSP_2	9	.325	.393

APPENDIX C - PAIRED SAMPLES T-TEST RESULTS FOR CONTROL GROUP

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
					Upper	Lower			
Pair 1	Syst BP - Syst BP_2	-1.778	3.232	1.077	-4.262	.706	-1.650	8	.137
Pair 2	Diast BP - Diast BP_2	-1.111	2.713	.904	-3.197	.974	-1.229	8	.254
Pair 3	PP - PP_2	-.667	4.583	1.528	-4.189	2.856	-.436	8	.674
Pair 4	MAP - MAP_2	-1.69778	2.72843	.90948	-3.79504	.39948	-1.867	8	.099
Pair 5	wh ratio - wh ratio_2	-.0032270	.0307979	.0102660	-.0269004	.0204464	-.314	8	.761
Pair 6	bodyfat - bodyfat_2	.5556	4.5302	1.5101	-2.9267	4.0378	.368	8	.722
Pair 7	BMI - BMI_2	.0333	1.8446	.6149	-1.3845	1.4512	.054	8	.958
Pair 8	tc - tc_2	-6.444	35.553	11.851	-33.773	20.884	-.544	8	.601
Pair 9	hdl - hdl_2	1.333	12.083	4.028	-7.955	10.621	.331	8	.749
Pair 10	trg - trg_2	2.667	109.843	36.614	-81.766	87.100	.073	8	.944
Pair 11	ldl - ldl_2	-20.857	22.711	8.584	-41.862	.147	-2.430	6	.051
Pair 12	nhdl - nhdl_2	-8.000	45.774	15.258	-43.185	27.185	-.524	8	.614
Pair 13	tc_hdl - tc_hdl_2	-.2222	1.8185	.6062	-1.6200	1.1756	-.367	8	.723
Pair 14	glumg - glumg_2	-.889	10.289	3.430	-8.798	7.020	-.259	8	.802
Pair 15	HR bpm - HR bpm_2	-3.778	19.169	6.390	-18.512	10.957	-.591	8	.571
Pair 16	time_mins - time_mins_2	.00333	1.00906	.33635	-.77230	.77896	.010	8	.992
Pair 17	VO2 Max - VO2 Max_2	.222	3.153	1.051	-2.202	2.646	.211	8	.838
Pair 18	ECW_TBW - ECW_TBW_2	-.000333	.002121	.000707	-.001964	.001297	-.471	8	.650
Pair 19	BMR - BMR_2	-8.222	25.148	8.383	-27.553	11.109	-.981	8	.355
Pair 20	FSP - FSP_2	.000	3.428	1.143	-2.635	2.635	.000	8	1.000

APPENDIX D - STATISTICS FOR EXPERIMENTAL GROUP

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	Syst BP	131.08	12	8.129	2.347
	Syst BP_2	129.50	12	7.514	2.169
Pair 2	Diast BP	84.00	12	6.252	1.805
	Diast BP_2	82.83	12	5.813	1.678
Pair 3	PP	47.08	12	4.502	1.300
	PP_2	46.67	12	6.853	1.978
Pair 4	MAP	127.2575	12	8.62017	2.48843
	MAP_2	125.5683	12	7.36314	2.12555
Pair 5	wh ratio	.950297	12	.0401671	.0115952
	wh ratio_2	.944891	12	.0390546	.0112741
Pair 6	Bodyfat	28.050	12	8.9280	2.5773
	bodyfat_2	26.850	12	8.6567	2.4990
Pair 7	BMI	29.150	12	4.8812	1.4091
	BMI_2	28.858	12	4.1369	1.1942
Pair 8	Tc	181.25	12	30.242	8.730
	tc_2	178.67	12	16.876	4.872
Pair 9	Hdl	52.42	12	15.204	4.389
	hdl_2	45.17	12	14.911	4.304
Pair 10	Trg	100.33	12	67.834	19.582
	trg_2	114.75	12	52.963	15.289
Pair 11	Ldl	110.22	9	31.148	10.383
	ldl_2	112.33	9	21.319	7.106
Pair 12	Nhdl	129.75	12	35.162	10.150
	nhdl_2	133.58	12	22.187	6.405
Pair 13	tc_hdl	3.825	12	1.4907	.4303
	tc_hdl_2	4.450	12	1.7558	.5068
Pair 14	Glumg	92.50	12	16.600	4.792
	glumg_2	93.25	12	14.747	4.257
Pair 15	HR bpm	123.67	12	11.380	3.285
	HR bpm_2	120.83	12	15.845	4.574
Pair 16	time_mins	15.3633	12	1.21918	.35195
	time_mins_2	14.4267	12	1.41902	.40963
Pair 17	VO2 Max	37.00	12	4.592	1.326
	VO2 Max_2	40.83	12	4.970	1.435
Pair 18	ECW_TBW	.41642	12	.128169	.036999
	ECW_TBW_2	.37792	12	.005728	.001654
Pair 19	BMR	1754.75	12	122.224	35.283
	BMR_2	1766.33	12	121.248	35.001
Pair 20	FPS	1.42	12	3.147	.908
	FPS_2	2.67	12	2.570	.742

APPENDIX E - PAIRED SAMPLES CORRELATIONS FOR EXPERIMENTAL GROUP

		N	Correlation	Sig.
Pair 1	Syst BP & Syst BP_2	12	.693	.012
Pair 2	Diast BP & Diast BP_2	12	.810	.001
Pair 3	PP & PP_2	12	.042	.896
Pair 4	MAP & MAP_2	12	.903	.000
Pair 5	wh ratio & wh ratio_2	12	.952	.000
Pair 6	Bodyfat & bodyfat_2	12	.978	.000
Pair 7	BMI & BMI_2	12	.986	.000
Pair 8	tc & tc_2	12	.604	.038
Pair 9	hdl & hdl_2	12	.788	.002
Pair 10	trg & trg_2	12	.745	.005
Pair 11	ldl & ldl_2	9	.736	.024
Pair 12	nhdl & nhdl_2	12	.778	.003
Pair 13	tc_hdl & tc_hdl_2	12	.781	.003
Pair 14	glumg & glumg_2	12	.014	.967
Pair 15	HR bpm & HR bpm_2	12	.327	.299
Pair 16	time_mins & time_mins_2	12	.540	.070
Pair 17	VO2 Max & VO2 Max_2	12	.717	.009
Pair 18	ECW_TBW & ECW_TBW_2	12	.152	.637
Pair 19	BMR & BMR_2	12	.990	.000
Pair 20	FPS & FPS_2	12	.839	.001

APPENDIX F - PAIRED SAMPLES T-TEST RESULTS FOR EXPERIMENTAL GROUPS

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
					Upper	Lower			
Pair 1	Syst BP - Syst BP_2	1.583	6.156	1.777	-2.328	5.495	.891	11	.392
Pair 2	Diast BP - Diast BP_2	1.167	3.738	1.079	-1.208	3.541	1.081	11	.303
Pair 3	PP - PP_2	.417	8.039	2.321	-4.691	5.525	.180	11	.861
Pair 4	MAP - MAP_2	1.68917	3.72655	1.07576	-.67857	4.05690	1.570	11	.145
Pair 5	wh ratio - wh ratio_2	.0054062	.0122911	.0035481	-.0024031	.0132156	1.524	11	.156
Pair 6	bodyfat - bodyfat_2	1.2000	1.8825	.5434	.0039	2.3961	2.208	11	.049*
Pair 7	BMI - BMI_2	.2917	1.0561	.3049	-.3794	.9627	.957	11	.359
Pair 8	tc - tc_2	2.583	24.141	6.969	-12.755	17.922	.371	11	.718
Pair 9	hdl - hdl_2	7.250	9.799	2.829	1.024	13.476	2.563	11	.026*
Pair 10	trg - trg_2	-14.417	45.348	13.091	-43.229	14.396	-1.101	11	.294
Pair 11	ldl - ldl_2	-2.111	21.157	7.052	-18.374	14.151	-.299	8	.772
Pair 12	nhdl - nhdl_2	-3.833	22.687	6.549	-18.248	10.581	-.585	11	.570
Pair 13	tc_hdl - tc_hdl_2	-.6250	1.1038	.3186	-1.3263	.0763	-1.961	11	.076
Pair 14	glumg - glumg_2	-.750	22.054	6.366	-14.763	13.263	-.118	11	.908
Pair 15	HR bpm - HR bpm_2	2.833	16.202	4.677	-7.461	13.128	.606	11	.557
Pair 16	time_mins - time_mins_2	.93667	1.27746	.36877	.12500	1.74833	2.540	11	.027*
Pair 17	VO2 Max - VO2 Max_2	-3.833	3.614	1.043	-6.130	-1.537	-3.674	11	.004*
Pair 18	ECW_TBW -ECW_TBW_2	.038500	.127422	.036784	-.042460	.119460	1.047	11	.318
Pair 19	BMR - BMR_2	-11.583	16.994	4.906	-22.381	-.786	-2.361	11	.038
Pair 20	FPS - FPS_2	-1.250	1.712	.494	-2.338	-.162	-2.529	11	.028

Note: significant differences are highlighted and marked with *

APPENDIX G - TESTING FOR EQUALITY OF MEAN VALUES BETWEEN CONTROL AND EXPERIMENTAL GROUPS

	Group	N	Mean	Std. Deviation	Std. Error Mean
Bodyfat	Experimental	12	28.050	8.9280	2.5773
	Control	9	22.722	4.0993	1.3664
Hdl	Experimental	12	52.42	15.204	4.389
	Control	9	49.67	10.920	3.640
time_mins	Experimental	12	15.3633	1.21918	0.35195
	Control	9	15.0511	1.52194	0.50731
VO2 Max	Experimental	12	37.00	4.592	1.326
	Control	9	40.56	3.087	1.029
BMR	Experimental	12	1754.75	122.224	35.283
	Control	9	1786.56	163.847	54.616
FPS	Experimental	12	1.42	3.147	0.908
	Control	9	1.78	3.073	1.024

APPENDIX H - Independent Samples Test

		Levene's Test for Equality of Variances		t-test for Equality of Means						
									95% Confidence Interval of the Difference	
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	Upper	Lower
Bodyfat	Equal variances assumed	4.258	0.053	1.656	19	.114	5.3278	3.2170	-1.4055	12.0610
Hdl	Equal variances assumed	2.655	0.120	0.460	19	.651	2.750	5.982	-9.771	15.271
time_mins	Equal variances assumed	0.371	0.550	0.523	19	.607	.31222	0.59747	-.93829	1.56274
VO2 Max	Equal variances assumed	0.427	0.521	-2.002	19	.060	-3.556	1.776	-7.273	0.162
BMR	Equal variances assumed	0.538	0.472	-0.511	19	.615	-31.806	62.286	-162.173	98.561
FPS	Equal variances assumed	0.073	0.790	-0.263	19	.796	-.361	1.374	-3.237	2.515

APPENDIX I - EFFECT SIZE

- Variable	Units	T-value	Cohen's D
Syst BP	Systolic blood pressure (mm/hg)	0.891	0.38
Diast BP	Diastolic blood pressure (mm/hg)	1.081	0.46
PP	Partial blood pressure (mm/hg)	0.18	0.08
MAP	Mean arterial pressure (mm/hg)	1.57	0.67
wh ratio	Waist-to-Hip ratio	1.524	0.65
bodyfat	Bodyfat percentage	2.208	
BMI	BMI (kg/m2)	0.957	0.41
tc	Total cholesterol (mg/dl)	0.371	0.16
hdl	High-density lipoprotein (mg/dl)	2.563	
trg	Triglycerides (mg/dl)	-1.101	0.47
ldl	Low-density lipoprotein (mg/dl)	-0.299	0.13
nhdl	Non-high density lipoprotein (mg/dl)	-0.585	0.25
tc_hdl	Total cholesterol/High-density lipoprotein ratio	-1.961	
glumg	Glucose (mg/dl)	-0.118	0.05
HR bpm	Heart rate (bpm)	0.606	0.26
time_mins	Time in minutes	2.54	
VO2 Max	VO2 Max capacity (ml/min/kg)	-3.674	
ECW_TBW	Extracellular to Total Body Water (ECW/TBW) ratio	1.047	0.45
BMR	Basal Metabolic rate (kcal per day)	-2.361	
FPS	Framingham Point Scores	-2.529	
LBM	Lean Body Mass (kg)	-1.037	0.44

APPENDIX J - ESTIMATE OF 10-YEAR RISK FOR CORONARY HEART DISEASE FOR MEN (FRAMINGHAM POINT SCORES)

Age (years)	FPS
30-34	-1
35-39	0
40-44	1
45-49	2
50-54	3
55-59	4
60-64	5
65-69	6
70-74	7

Total Cholesterol (mg/dl)	FPS
<160	-3
160-199	0
200-239	1
240-279	2
>/=280	3

HDL Cholesterol (mg/dl)	FPS
<35	2
35-44	1
45-49	0
50-59	0
>/=60	-2

Blood Pressure					
Systolic (mmHg)	Diastolic (mmHg)				
	<80	80-84	85-89	90-99	>100
<120	0 pts				
120-129		0 pts			
130-139			1 pts		
140-159				2 pts	
≥160					3 pts

Diabetes (Glucose above 140 mg/dl)	
	FPS
No	0
Yes	2

Source: http://www.medicalcriteria.com/criteria/car_framingham_score_men.htm

APPENDIX K - SUMMARY STATISTICS FOR EXPERIMENTAL GROUP INCLUDING T-TEST RESULTS

	Pre-test	Post-test	Difference	t-value	Significance (p-value)
Syst BP	131.08 (8.129)	129.50 (7.514)	1.583 (6.156)	0.891	0.392
Diast BP	84.00 (6.252)	82.83 (5.813)	1.167 (3.738)	1.081	0.303
PP	47.08 (4.502)	46.67 (6.853)	0.417 (8.039)	0.180	0.861
MAP	127.2575 (8.62017)	125.5683 (7.36314)	1.68917 (3.72655)	1.570	0.145
wh ratio	.950297 (.0401671)	.944891 (.0390546)	0.0054 (0.0123)	1.524	0.156
bodyfat	28.050 (8.9280)	26.850 (8.6567)	1.2 (1.8825)	2.208	0.049*
BMI	29.150 (4.8812)	28.858 (4.1369)	0.2917 (1.0561)	0.957	0.359
tc	181.25 (30.242)	178.67 (16.876)	2.583 (24.141)	0.371	0.718
hdl	52.42 (15.204)	45.17 (14.911)	7.25 (9.799)	2.563	0.026*
trg	100.33 (67.834)	114.75 (52.963)	-14.417 (45.348)	-1.101	0.294
ldl	110.22 (31.148)	112.33 (21.319)	-2.111 (21.157)	-0.299	0.772
nhdl	129.75 (35.162)	133.58 (22.187)	-3.833 (22.687)	-0.585	0.570
tc_hdl	3.825 (1.4907)	4.450 (1.7558)	-0.625 (1.1038)	-1.961	0.076
glumg	92.50 (16.600)	93.25 (14.747)	-0.75 (22.054)	-0.118	0.908
HR bpm	123.67 (11.380)	120.83 (15.845)	2.833 (16.202)	0.606	0.557
time_mins	15.3633 (1.21918)	14.4267 (1.41902)	0.937 (1.278)	2.540	0.027*
VO2 Max	37.00 (4.592)	40.83 (4.970)	-3.833 (3.614)	-3.674	0.004*
ECW_TBW	0.41642 (0.128169)	.37792 (.005728)	0.0385 (0.127)	1.047	0.318
BMR	1754.75 (122.224)	1766.33 (121.248)	-11.583 (16.994)	-2.361	0.038
FPS	1.42 (3.147)	2.67 (2.570)	-1.25 (1.712)	-2.529	0.028

Note: mean values are shows with standard deviation in brackets.

* p-value is below level of significance 0.05 (the row is highlighted for significant difference)

APPENDIX L - SUMMARY STATISTICS FOR CONTROL AND EXPERIMENTAL GROUPS

Variable	Description	experimental group			control group		
		pre-test	post-test	standard error of the difference	pre-test	post-test	standard error of the difference
Syst BP	Systolic blood pressure (mm/hg)	131.08	129.5	1.777	123.33	125.11	1.077
Diast BP	Diastolic blood pressure (mm/hg)	84	82.83	1.079	81.33	82.44	0.904
PP	Partial blood pressure (mm/hg)	47.08	46.67	2.321	42	42.67	1.528
MAP	Mean arterial pressure (mm/hg)	127.2575	125.5683	1.07576	122.0333	123.7311	0.90948
wh ratio	Waist-to-Hip ratio	0.950297	0.944891	0.0035481	0.929671	0.932898	0.010266
bodyfat	Bodyfat percentage	28.05	26.85	0.5434	22.722	22.167	1.5101
BMI	BMI (kg/m2)	29.15	28.858	0.3049	26.256	26.222	0.6149
tc	Total cholesterol (mg/dl)	181.25	178.67	6.969	190.67	197.11	11.851
hdl	High-density lipoprotein (mg/dl)	52.42	45.17	2.829	49.67	48.33	4.028
trg	Triglycerides (mg/dl)	100.33	114.75	13.091	140	137.33	36.614
ldl	Low-density lipoprotein (mg/dl)	110.22	112.33	7.052	106.14	127	8.584
nhdl	Non-high density lipoprotein (mg/dl)	129.75	133.58	6.549	140.89	148.89	15.258
tc_hdl	Total cholesterol/High-density lipoprotein ratio	3.825	4.45	0.3186	4	4.222	0.6062
glumg	Glucose (mg/dl)	92.5	93.25	6.366	92.33	93.22	3.43
HR bpm	Heart rate (bpm)	123.67	120.83	4.677	118.56	122.33	6.39
time_mins	Time in minutes	15.3633	14.4267	0.36877	15.0511	15.0478	0.33635
VO2 Max	VO2 Max capacity (ml/min/kg)	37	40.83	1.043	40.56	40.33	1.051
ECW_TBW	Extracellular to Total Body Water (ECW/TBW) ratio	0.41642	0.37792	0.036784	0.37478	0.37511	0.000707
BMR	Basal Metabolic rate (kcal per day)	1754.75	1766.33	4.906	1786.56	1794.78	8.383
FPS	Framingham Point Scores	1.42	2.67	0.494	1.78	1.78	1.143

APPENDIX M - GAME PARTICIPATION

	Games															
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
Anderson	X		X	X	X	X	X	X	X	X	X	X	X	X	X	X
Bartley, P	X	X	X	X		X	X	X	X	X	X	X	X	X	X	X
Brgan M	X	X		X	X	X	X	X	X	X	X	X	X	X	X	X
Chapman S	X	X	X	X	X	X	X	X	X	X	X		X	X	X	X
FordhamR	X	X	X	X	X	X	X		X	X	X	X	X	X	X	X
Hilebrand	X	X	X	X	X	X	X	X	X		X	X	X	X	X	X
Iamello R.	X	X	X	X	X	X	X	X	X	X	X		X	X	X	X
Kalbfish C	X	X	X	X	X		X	X	X	X	X	X	X	X	X	X
Kalinovski	X	X	X	X	X	X	X	X		X	X	X	X	X	X	X
Moll H	X	X	X		X	X	X	X	X	X	X	X	X	X	X	X
Murray S	X	X	X	X	X	X	X	X	X	X	X	X		X	X	X
Sandro B	X	X	X	X	X	X	X	X	X	X	X	X	X	X		X
Coverley D	X			X	X	X						X				

Note: X represents game attendance